VOLUME 49 NUMBER 2

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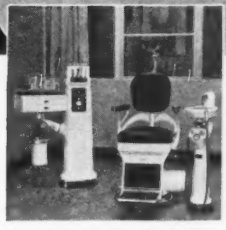

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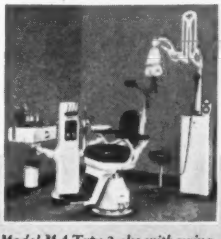

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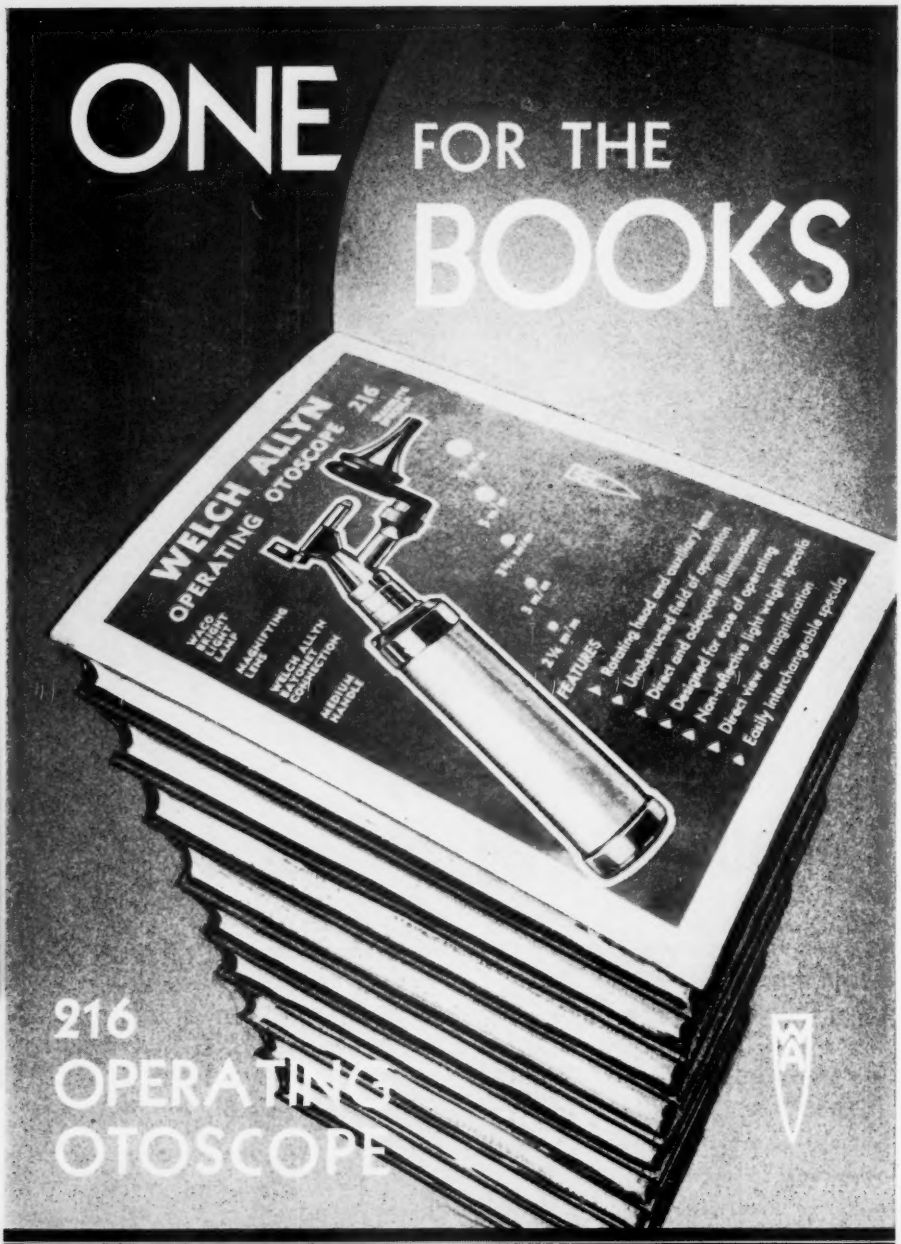

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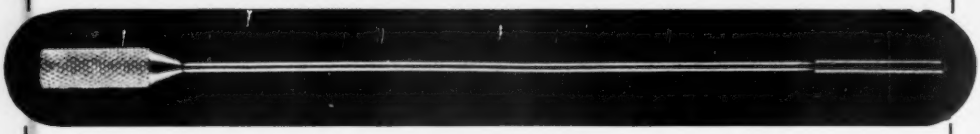

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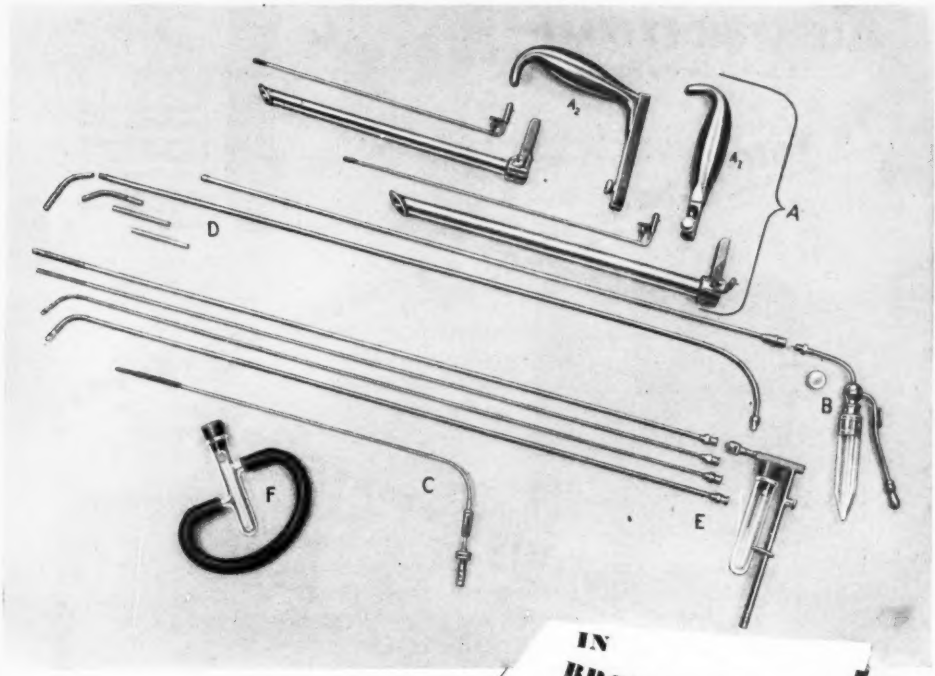

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ABSCESS OF THE FRONTAL LOBE SECONDARY TO ETHMOIDITIS

JOSEPH H. KLER, M.D.
NEW BRUNSWICK, N. J.

ABSCESS of the brain is a rare complication of sinusitis in general and an extremely rare complication of ethmoiditis in particular. In a series of 14,534 autopsies at the London Hospital, Evans [1] found 194 cases of brain abscess, and in only 14 of these was the condition rhinogenic. Onodi [2] reported 4 cases of rhinogenic brain abscess in a series of 13,400 autopsies, while Courville and Rosenvold [3] reported 2 cases of brain abscess in their series of 15,000 autopsies. The vast majority of these abscesses were due to infection of the frontal sinus. In Eagleton's [4] series of 104 abscesses of the frontal lobe, only 9 were due to infection limited to the ethmoid sinus. The author has found no record of brain abscess due to infection limited to the ethmoid sinus in the literature during the past seventeen years.

Sinusitis is a very common medical entity, and in my experience ethmoiditis is commoner than infection in the other paranasal sinuses. It seems rather strange that such a frequent infection in an area of the body that seems so vulnerable should be so relatively free of intracranial complications. All surveys of complications of ethmoidal infection corroborate Dreyfuss' [5] findings: (1) that fatal complications of ethmoiditis usually follow the acute form of the disease and that the ethmoid cells are seldom exclusively involved; (2) that infection usually travels by way of the lamina cribrosa to the intracranial space, and (3) that therefore septic meningitis is the commonest complication. Cour-

From the Department of Ophthalmology and Otolaryngology, St. Peter's General Hospital.

1. Evans, W.: Pathology and Aetiology of Brain Abscess, Lancet **1**:1231 (June 6); 1289 (June 13) 1931.

2. Onodi, A.: The Oculo-Orbital, Intra-Cranial and Cerebral Complications of Diseases of the Nasal Accessory Sinuses, Laryngoscope **19**:801 (Nov.) 1909.

3. Courville, C. B., and Rosenvold, L. K.: Intracranial Complications of Infections of Nasal Cavities and Accessory Sinuses: Survey of Lesions Observed in Fifteen Thousand Autopsies, Arch. Otolaryng. **27**:692 (June) 1938.

4. Eagleton, W. P.: Brain Abscess: Its Surgical Pathology and Operative Technic, New York, The Macmillan Company, 1922, pp. 23 and 249.

5. Dreyfuss, R.: Zur pathologischen Anatomie der Brustdrüse: Die Krankheiten des Gehirns und seiner Adnepa in Gefolge von Naseneiterungen, Jena, G. Fischer, 1896, pp. 45 and 46.

ville and Rosenvold, in their exhaustive study, found thrombosis of the cavernous sinus the second most frequent complication. McKinney [6] reported a fatal case of brain abscess following osteomyelitis of the frontal bone due to an original infection limited to the ethmoid sinus. Rugg Gunn and Suggitt [7] reported many more intracranial complications of acute sinusitis than of chronic sinusitis. In Maxwell's [8] series of 1,268 cases of acute and chronic sinusitis there were 22 deaths due to complications of the sinusitis, and of these 3 were due to ethmoiditis.

There must be a logical explanation for this unusual circumstance. It must be agreed that the location of the ethmoid sinuses is an extremely vulnerable one—separated from the brain, orbit and other sinuses only by thin bony walls. The anatomic location of the ethmoid sinus, therefore, does not explain the infrequency of intracranial complications of ethmoidal infection. The explanation must be in the intrinsic structure of the ethmoid sinuses together with the nature of their blood supply.

Van Alyea's [9] classic description of the ethmoid labyrinth bears repeating (in part) because it throws much light on this subject. He found greater variability in the anatomic characteristics of the ethmoid sinus than in the other sinuses, and he also found more pathologic change in the mucosa of the unusually large and variable ethmoid cells. According to his description, the ethmoid labyrinth is divided by various bony lamellas, and the ground plate of the middle turbinate divides the labyrinth into the two major sections, the anterior and the posterior. He stated:

> These two major groups of cells are further divided by the lamellas of the remaining turbinates and accessory turbinate. [The lamellas normally prevent communication between groups of cells.] Ethmoid cells commonly drain into grooves. These form fairly parallel lines along the face of the upper lateral nasal wall. They are the hiatus semilunaris and the bullar ostium in the middle meatus, the posterior groove in the superior meatus and the postreme groove in the supreme meatus. One groove may accommodate the ostiums of several cells or may be the wide drainage space of 1 cavity. Occasionally the ostiums of cells are located outside the normal drainage groove, and this tends to isolate these cells from the others of a group. Cells draining into a common area should be similarly involved in an infective process. It is for this reason that

6. McKinney, R.: Osteomyelitis of the Frontal Bone, Arch. Otolaryng. **28**:1 (July) 1938.

7. Rugg Gunn, M. A., and Suggitt, S. C.: Brain Abscess, J. Laryng. & Otol. **57**:55 (Feb.) 1942.

8. Maxwell, D.: Analysis of the Statistical Tables for the Years 1907 to 1926 Inclusive, Covering a Period of Twenty Years, J. Laryng. & Otol. **44**:105 (Feb.) 1929.

9. Van Alyea, O. E.: Ethmoid Labyrinth: Anatomic Study with Consideration of the Clinical Significance of Its Structural Characteristics, Arch. Otolaryng. **29**:881 (June) 1939.

the maxillary sinus, the infundibular cells and occasionally the frontal sinus are simultaneously infected. For the same reason bullar cells, which have a separate drainage ostium, often escape an infection involving all other anterior cavities.

Although the bony structure of the ethmoid labyrinth is thin, the bone is of a compact, dense lamellar type. The blood supply of the ethmoid bone is by way of the ethmoid branches of the ophthalmic artery, and there are rich anastomoses with surrounding arteries. The venous drainage is also complex, being principally into the ethmoidal veins, which empty into the ophthalmic veins. These veins communicate with those of the dura and the superior sagittal sinus. Schaeffer [10] also described an anterior ethmoidal vein which passes through the cribriform plate and connects with the venous plexus of the olfactory bulb and the frontal lobe of the brain. Kramer and Som [11] found that the venules of the mucoperiosteum communicate with the large cranial sinuses and with the veins of the dura. These perforating venules are surrounded by perivascular spaces, which are probably lymph channels communicating with the intracranial contents. The lymphatic drainage of the sinuses is principally into the retropharyngeal nodes. The subdural space communicates indirectly with the extracranial lymphatics and directly with the perineural spaces of the olfactory nerve filaments. Thus there is a complex structure completely encased by dense lamellar bone with few dehiscences, with an unusually rich blood supply and with a lymphatic drainage principally away from the intracranial contents.

This anatomic arrangement is exceptionally good for the prevention of spread of the usual low grade infections. The cells of the ethmoid labyrinth are arranged in groups, each having separate ostiums for drainage. Even though the septal walls are thin, the bone resists infection, and the infective process in any group of cells is kept reasonably isolated to permit of its control by the natural defense mechanism of the body. In an acute and virulent infection this mechanism is not adequate. Kramer and Som were impressed with the formation of submucosal abscesses. At the site of the submucosal abscess there is then developed an osteitis with eventual necrosis of the bone. Since the interseptal walls are the thinnest, they will break down first, and there is developed an empyema of the sinus. The coalescence of cells can be followed by roentgenologic studies. The lamina papyracea is the next vulnerable wall, and when it is destroyed the ethmoidal infection produces a subperiosteal abscess of the orbit. If the acute infection is not

10. Schaeffer, J. P.: The Nose, Paranasal Sinuses, Nasolacrimal Passageways and Olfactory Organ in Man, Philadelphia, P. Blakiston's Sons & Co., 1930.

11. Kramer, R., and Som, M. L.: Intracranial Pathways of Infection from Diseases of the Sphenoid and Ethmoid Sinuses, Arch. Otolaryng. **32**:744 (Oct.) 1940.

controlled it develops into meningitis or thrombosis of the cavernous sinus. However, destruction of the roof of the ethmoid sinus can produce localized meningitis, followed by the development of a subcortical abscess. I report here a case illustrating this complication.

It is my belief that chronic infection in the ethmoid sinuses is the principal cause of brain abscess of ethmoid origin. The extension of the infection is by contiguity, either by osteitis, causing local necrosis with subsequent local meningitis, encephalitis and formation of subcortical abscess, or by thrombophlebitis of any of the veins communicating with the dura. There seems to be no uniformity in the type of infecting organism. The bacteria reach the subcortical tissues and set up the usual reaction to inflammation. Its extent depending on the virulence of the organism and the resistance of the body, an attempt is made by the brain tissue to wall off the infection. This walling off is the formation of a capsule. A capsule forms in about three to four weeks according to Alpers,[12] although Kaplan[13] reported a case in which a capsule formed only after six weeks. If the path of invasion remains it is the classic "stalk." There is much difference of opinion on the presence of a stalk in abscess of the frontal lobe in general. Eagleton did not find a stalk frequently, but Atkinson[14] stated that he found a point of entry on the dura in all cases of adjacent abscess of the brain.

The symptoms of brain abscess depend on the stage of development and the location of the infection. Craig and Adson[15] stated the belief that meningitis, encephalitis and abscess of the brain are merely stages of development of the process. Specifically, the clinical picture presented by patients with brain abscess depends on the following factors: (1) the presence of infection, (2) the presence of an intracranial mass giving rise to increased intracranial pressure and (3) localizing symptoms referable to the portion of the brain involved. Any or all of these manifestations may be present, either alone or in any combination. It is not the purpose of this paper to review all known symptoms but, rather, to present the clinical picture of the 3 cases of abscess of the frontal lobe of ethmoid origin which I have encountered and to emphasize the lessons I have learned, in the hope that they will be of some value to others.

REPORT OF CASES

Acute Abscess of the Frontal Lobe.—This type of abscess is the result of an acute and a virulent infection to which the patient has not

12. Alpers, B. J.: Abscess of the Brain: Relation of the Histologic to the Clinical Features, Arch. Otolaryng. **29**:199 (Feb.) 1939.

13. Kaplan, A.: Abscess of the Brain, Arch. Otolaryng. **29**:385 (April) 1935.

14. Atkinson, M.: Otogenous Cerebellar Abscess, Ann. Otol., Rhin. & Laryng. **47**:1020 (Dec.) 1938.

15. Craig, W. M., and Adson, A. W.: Abscess of the Brain, S. Clin. North America **17**:1077 (Aug.) 1937.

been able to develop an adequate resistance. It is found as a complication of acute ethmoiditis as a rule. This condition is well illustrated by the following case which I observed.

CASE 1.—A 5 year old child, critically ill, was first seen by me on the day of admission to the hospital. There were typical findings of acute ethmoiditis and periorbital abscess together with nuchal rigidity. The diagnosis of ethmoidal empyema and periorbital abscess was immediately made, and because of the seriousness of the child's condition it was decided to drain the orbital and the sinal infection at once and then determine the exact nature of the intracranial complication. The orbital abscess was drained through an external incision, and the ethmoid sinus was drained intranasally. Both the orbital and the sinal abscess were extremely large, and when the pus was cleaned away the roof of the ethmoid sinus was found to be necrotic. After careful removal of the necrotic bone, an extradural abscess was entered, and a fistulous tract to a subcortical abscess was found. The infections, although extensive, were well walled off. Drainage and chemotherapy resolved the condition successfully.

The diagnosis of brain abscess was presumptive, of course; but in general the determining factors were the history of an infection of the upper respiratory tract, followed by the obvious presence of a sinal infection, a periorbital abscess and signs of meningeal irritation and increased intracranial pressure. As Grant [16] stated, such a chain of events makes the diagnosis virtually inescapable. The treatment of acute abscess of the brain is much more difficult than making the diagnosis. Present methods of treatment of sinusitis have reduced the incidence of acute abscess of the brain, as has been observed by Davidoff.[17] The sinal infection, and the orbital infection, if present, must be treated first. Chemotherapy and appropriate drainage are necessary. In Grant's [16] opinion the primary focus of a suspected adjacent abscess should be eradicated before the abscess is drained—first, because the operation may show the area in which the infection has passed through the bone to the dura and thus help in suggesting the position of the abscess and, second, because, unless the primary focus has been eliminated, reinfection of the cavity of the abscess may occur and nullify an apparently successful drainage.

Chronic Abscess of the Frontal Lobe.—This type of abscess is as a rule an insidious, asymptomatic condition that can easily progress to a critical stage before the patient seeks medical attention. The following 2 cases well illustrate the problem encountered.

CASE 2.—A man had a history of a low grade chronic ethmoiditis for many years. During inclement weather he would complain of increased nasal and

16. Grant, F. C.: Brain Abscess: Collective Review, Internat. Abstr. Surg. **72**:118, 1941; in Surg., Gynec. & Obst., February 1941.

17. Davidoff, L. M.: Brain Abscess, Clinics **4**:343 (Aug.) 1945.

postnasal discharge and headaches but did not seek medical attention during the ten years prior to the development of intracranial symptoms. He worked up to the day before admission to the hospital. His only complaint was an unbearable headache of about three weeks' duration. On examination he did not appear critically ill. There was pus in his right nostril, and a roentgenogram of the sinuses revealed cloudy ethmoid cells on the right. There was papilledema of about 3 D. in both eyes. These examinations were performed on the first day of admission. The following day the patient had an unexpected crisis: He became unconscious; both eyes deviated to the left, and there were nuchal rigidity and swelling of the right upper eyelid. It was then quite evident that he had a brain abscess which had ruptured into the lateral ventricle. Although the patient was moribund, it was decided to explore the swelling of the right upper eyelid through a supraorbital incision. The floor of the frontal sinus and the roof of the ethmoid sinus were necrotic, and both sinuses contained pus. The posterior

Fig. 1.—Roentgenogram of the sinuses.

wall of the frontal sinus was also necrotic, and on its removal an extradural abscess was revealed. This abscess had a sinus that apparently led to a subcortical abscess. The patient died the following day.

There are several lessons in this case, and the first and foremost is for one not to permit either the physician or the patient to regard chronic ethmoiditis lightly. It is a potential danger that must be guarded against. The second lesson is that diagnosis of chronic encapsulated abscess of the brain in the presence of a low grade infection in the ethmoid sinuses is difficult.

Case 3.—The patient was an intelligent man, and one who would not ignore evidence of illness. One day he reported to the medical department of his employer complaining of periodic blurring of vision and slight weakness of his "knee." The nurse referred him to me for an examination of his eyes.

The examination revealed papilledema of 6 D. in the right eye and of 5 D. in the left eye. There was no peripheral field defect, although he had a large central scotoma, as would be expected. There were no other ocular signs. The left nostril had a few crusts of mucopus, and there was questionable anosmia in the nostril. A roentgenogram of the sinuses revealed only cloudiness in the ethmoid sinuses on the left side (fig. 1) and a roentgenogram of the skull indicated erosion about the sella, but the picture did not permit a definite diagnosis

Fig. 2.—Roentgenogram of the skull and the sinuses.

(fig. 3). The patient also gave a history of having had sinal infection about five years previously, although he had not been conscious of any symptoms since that time. These findings did not warrant any diagnosis other than an intracranial mass. The patient was referred to Dr. T. S. P. Fitch for neurologic consultation. Dr. Fitch found no focal signs on examination. However, the electroencephalogram indicated a brain abscess in the left frontal lobe (fig. 4). At operation the diagnosis was confirmed. Dr. Fitch found an abscess containing 200 cc. of pus. The abscess had a fistulous tract leading to the left

ethmoid cells. The patient made a remarkable recovery, and a month later I performed an intranasal ethmoidectomy. He had an unusually large suprabullar cell which contained pus. I did not probe it to find whether there was a tract to the dura, but it was reasonable to suppose that this cell was the source of the infection because the other cells did not contain pus. At present this man is back at work and free from symptoms. His vision is 20/20, and there is no elevation of the disk margins. So far there has been no evidence of development of atrophy of the optic nerve. It seems inconceivable that a man could have a brain abscess containing 200 cc. of pus and live, let alone be free from symptoms. If I had not had this patient under my care, I think that I should be skeptical of the possibility of such an occurrence.

Fig. 3.—Roentgenogram of the skull.

There are several lessons to be learned from this case also. An abscess of the brain may develop in ten days,[18] but it is my belief that in cases originating from ethmoiditis the abscess is of long standing. Cavenagh[19] reported a similar observation. It is my opinion that the patient develops a reasonable immunity to his sinal infection. However, acute coryza or other infections of the upper respiratory tract introduce

18. Kleinfeld, L.: Abscess of Frontal Lobe Secondary to Frontal Sinusitis, J. Mt. Sinai Hosp. **9**:96 (July-Aug.) 1942.

19. Cavenagh, J. B.: A Case of Left Frontal Lobe Abscess Complicating Chronic Sinus Infection, J. Laryng. & Otol. **51**:239 (April) 1936.

new bacteria, and with each succeeding reinfection there is further destruction of the mucosa of the ethmoid cells. The observations of Kramer and Som [11] give a logical explanation of the formation of localized areas of osteitis and necrosis of the roof of the ethmoid sinus. Because the infection is not a very virulent one and because the area of perforation is small, the localized meningitis may be entirely asymptomatic as far as the patient is concerned. Because the submucosal abscess is encapsulated, it will feed infection through the break in its capsule at the point of the osteitis to the meninges, with resulting encephalitis and subsequent formation of a subcortical abscess. The low virulence of the bacteria favors the formation of a capsule about the brain abscess. When this is accomplished there is a low grade infection

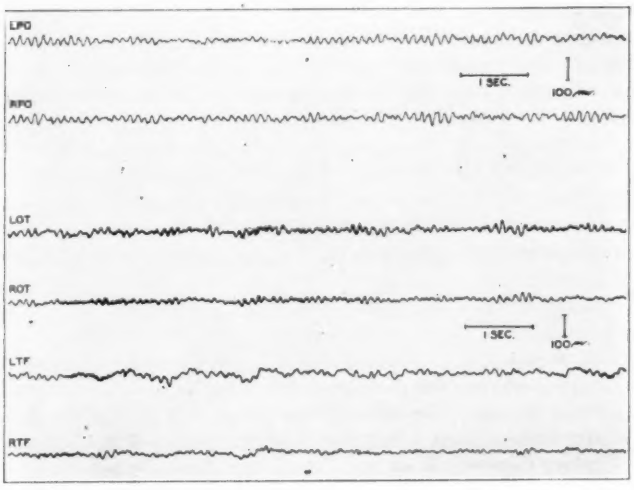

Fig. 4.—Electroencephalographic tracings. *LPO* indicates left parieto-occipital; *RPO*, right parieto-occipital; *LOT*, left occipitotemporal; *ROT*, right occipitotemporal; *LTF*, left temporofrontal, and *RTF*, right temporofrontal. The upper two tracings are from a normal subject and the others from the patient in case 3.

in the ethmoid sinus to which the patient has a reasonable tolerance and a walled-off infection within the brain substance which will give no septic symptoms or localizing symptoms in the early stages. Bertolet [20] reported a massive abscess of the frontal lobe that had many of the characteristics cited. Even though this condition is rare, I should like to emphasize the lessons these 2 cases have taught me. The diagnosis

20. Bertolet, J. A.: Atypical Massive Abscess of Frontal Lobe, Arch. Otolaryng. **28**:621 (Oct.) 1938.

is not easy. Smith [21] emphasized the value of the sedimentation test, but in the presence of a sinal infection this test is not significant. King [22] stressed the diagnostic importance of unilateral anosmia, but its presence is also difficult to interpret because many patients with chronic sinal infections have a reduced sense of smell. Kaplan [23] reported that encapsulated and uncomplicated abscesses of the brain frequently produce no signs of fever, leukocytosis or toxemia. Grant [16] emphasized the presence of papilledema in all cases of chronic encapsulated abscess. This has been the only constant finding in my cases. Headache is stressed by all authorities. It is my belief that in the type of abscess here described headache is a late symptom, and one that should be regarded as a danger signal. The electroencephalogram is an invaluable diagnostic aid. The cooperation of the neurosurgeon is essential. I fully realize that the treatment of abscess of the brain was pioneered by rhinologists, and I have the greatest respect for the late Dr. Eagleton and the many prominent rhinologists who followed him in the development of this field. However, I feel that the average rhinologist will do well to ask the aid of the neurosurgeon in both the diagnosis and the treatment of this baffling and obscure condition. This series of cases is too small for me to be dogmatic about conclusions, but the similarity of the cases is striking.

SUMMARY AND CONCLUSIONS

Brain abscess as a complication of acute and of chronic ethmoiditis is discussed.

An explanation of this condition on an anatomic and physiologic basis is made.

Complications of acute ethmoiditis are more frequent and more easily diagnosed than those of the chronic infection.

Chronic abscess of the brain as a complication of chronic ethmoiditis is an encapsulated abscess that gives few general or localizing signs.

Chronic abscesses of ethmoidal origin produce few symptoms and signs; papilledema was the most constant finding.

Electroencephalography is an invaluable aid in the diagnosis of this type of intracranial complication.

The assistance of the neurosurgeon in the management of these cases is essential.

151 Livingston Avenue.

21. Smith, G. W.: Clinical Observations on Twenty Proven Cases of Brain Abscess, U. S. Nav. M. Bull. **38**:358 (July) 1940.

22. King, J. E. J.: Symposium on Brain Abscess, Laryngoscope **50**:1146 (Dec.) 1940.

23. Kaplan, A.: Symposium on Brain Abscess, Laryngoscope **50**:1159 (Dec.) 1940.

SURGERY OF THE CHRONICALLY DISCHARGING EAR

HOWARD P. HOUSE, M.D.
LOS ANGELES

THE PURPOSE of this presentation is to stress certain important anatomic landmarks and surgical maneuvers which have proved extremely valuable to me in developing a technic of endaural temporal bone surgery.

The endaural incision is slightly changed from the original Lempert [1] technic in that the no. 1 incision (*A*) is placed more external in the skin of the ear canal. The no. 2 incision (*B*) is completed in the usual way, and the no. 3 incision (*C*) follows the edge of the conchal cartilage.

The incision is first outlined in gentian violet before local infiltration of the area with procaine hydrochloride obscures the usual landmarks. A Bard-Parker knife with a no. 15 blade is used to make the incision. A small tragus retractor replaces the Lempert ear speculum for this procedure. The triangular piece of skin removed is very small, and the entire incision is extracartilaginous. This incision allows greater preservation of tissue for later use as a skin flap in lining the mastoid cavity. This same endaural incision is used in all surgical procedures on the temporal bone.

After the self-retaining retractor is placed in position, a small elevator is used to elevate the periosteum over the zygomatic area. The triangular piece of fascia extending from the posterior membranous ear canal wall to the temporal muscle is then excised, care being taken to avoid the temporal muscle above and the superior membranous ear canal wall below. This procedure provides a more adequate exposure anteriorly toward the zygoma and eliminates the necessity of spreading the no. 2 endaural incision postoperatively in order to avoid atresia of the ear canal.

The horizontal canal has long been recognized as the most important landmark in temporal bone surgery. It is interesting to note that an

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From the Department of Otolaryngology, University of Southern California School of Medicine.

Read in part and illustrated by a motion picture before the Section on Laryngology, Otology and Rhinology at the Ninety-Seventh Annual Session of the American Medical Association, Chicago, June 27, 1948.

1. Lempert, J.: Endaural Antauricular Surgical Approach to the Temporal Bone: Principles Involved in This New Approach; Summary Report of 1,780 Cases, Arch. Otolaryng. **27**:555-587 (May) 1938.

imaginary line drawn directly posterior from the superior margin of the external bony ear canal will invariably bisect the horizontal canal deeper in the temporal bone. This is a great aid in visualizing the location of the antrum and aditus before any bone work is carried out. This valuable guide should be stressed in the teaching of surgical technic of the temporal bone.

The mastoid cortex and underlying cells are removed by the burr method. I have found it expedient to autoclave the entire dental arm together with the belt and the engine pulley. The sterilized engine pulley is replaced on the engine shaft by means of a sterile screw driver. More recently I have been wrapping the engine pulley in alcohol-soaked gauze, leaving it attached to the engine shaft. Repeated cultures have proved this technic to be sterile. This procedure eliminates the dental arm sleeve, which so frequently becomes entangled in the belt and pulley

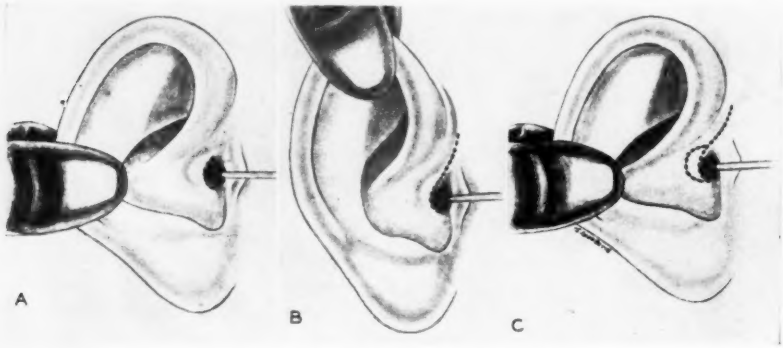

Fig. 1.—Endaural incision.

wheels. This method can be used only if the S. S. White Dental Arm is attached to the engine. This arm fits either the S. S. White or the Emesco Portable Dental Engine.

I use the perforating burr to enter the antrum. If the perforating burr is used properly, it will invariably enter the antrum without undue complications. The point of entrance of the perforating burr is such that an imaginary line drawn directly posterior from the superior margin of the external bony ear canal wall will cross the upper margin of the completed perforating burr hole. A line drawn directly superior from the posterior margin of the external bony ear canal wall will cross the anterior margin of the completed perforating burr hole.

The perforating burr must enter the bone at right angles to the patient's skull and must never penetrate the mastoid bone to a greater depth than that of the cutting portion of the burr. If these directions are invariably followed, one will not injure the dura superiorly, regardless of the amount of overhang, nor will one enter the lateral sinus regardless of its far anterior position.

The mastoid cells are exenterated by means of cutting burrs of various size. For reasons of safety, the nearer one is to the lateral sinus or dural plate the larger should be the burr. As these structures are approached there is a change in the sound of the burr, and slight bleeding may occur. A large burr is used to thin the posterior bony ear canal wall to a width of 1 or 2 mm. If the posterior bony ear canal

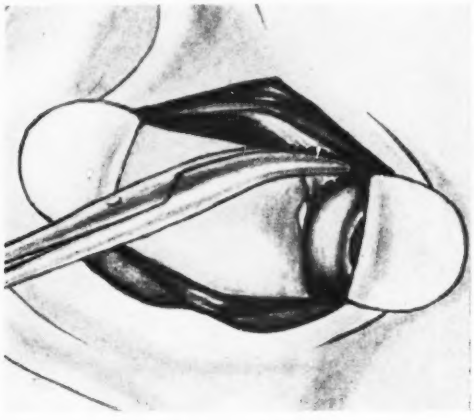

Fig. 2.—Triangular fascia grasped with hemostat and severed.

wall is adequately thinned and the dural plate is exposed directly superiorly, the posterior portion of the horizontal canal will be immediately visible and will invariably be bisected by the imaginary line drawn directly posterior from the superior margin of the external bony ear canal wall.

After the posterior bony ear canal wall has been adequately thinned, the dural plate exposed and the horizontal canal identified, a small burr is used to remove the zygomatic cells. These cells are removed from an external to internal approach and in a posterior to anterior direction between the dural plate superiorly and the superior margin of the external bony ear canal inferiorly. By this approach a definite shelf of thin, firm cortical bone will be exposed, which forms the external wall of the epitympanum. This landmark is constant in both wide and narrow

angle cases, and its importance has not been adequately stressed in the literature. The opening of the aditus is a triangle bounded by the dural plate superiorly, the bony shelf externally and the horizontal canal internally. If this shelf technic is followed, probing of the aditus, which may result in dislocation of the incus, is totally unnecessary. Exposure of the bony shelf completes the simple mastoidectomy, if one chooses to perform this procedure endaurally.

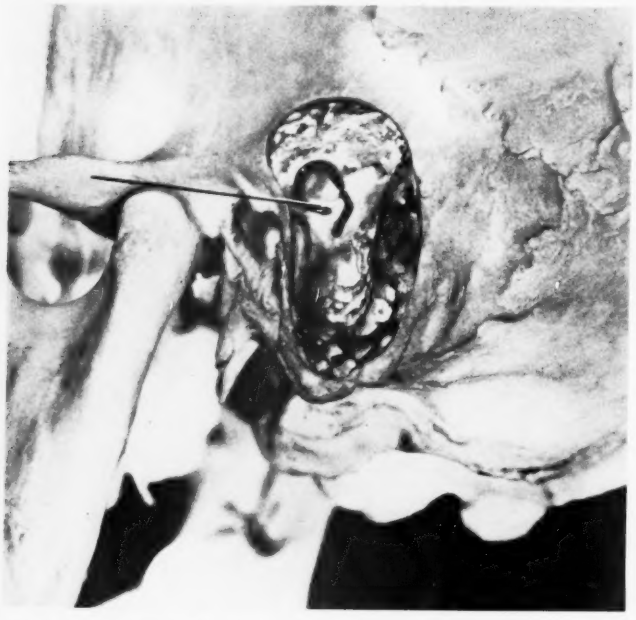

Fig. 3.—Note that a line drawn posteriorly from the superior margin of the bony canal bisects the horizontal canal deeper in the temporal bone.

MODIFIED RADICAL MASTOIDECTOMY

This operation is a continuation of the simple mastoid procedure just described. The thin bony shelf overlying the epitympanum is carefully removed with the use of a small curet. The removal is begun superiorly near the dural plate. In this way the short crus of the incus which lies just under the inferior margin of the bony shelf is not disturbed. Complete exposure of the entire short crus of the incus and malleus head and neck denotes an adequately thinned posterior-superior bony ear canal wall and bridge. These structures, therefore, should

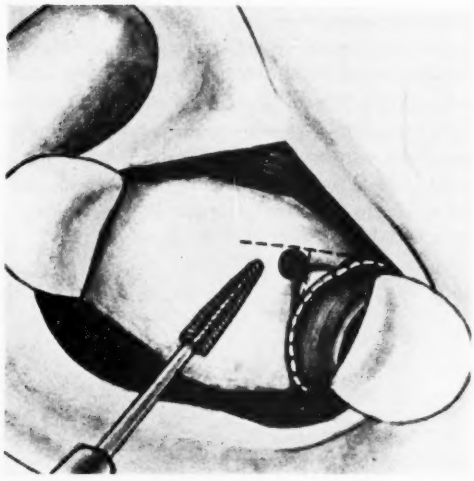

Fig. 4.—Note the exact position of the perforating burr hole in relation to the external bony ear canal wall.

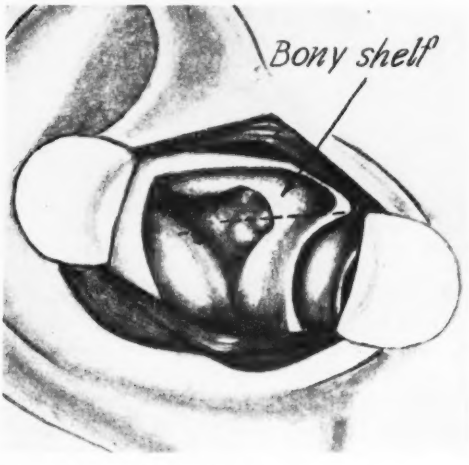

Fig. 5.—Note the bony shelf overlying the epitympanum (imaginary line bisects horizontal canal).

be visualized before the posterior-superior bony ear canal wall is taken down. In cases in which there is an extremely narrow angle it may be necessary to remove the posterior-superior bony ear canal wall before complete exposure of the incomalleolar joint can be accomplished.

The membranous ear canal is separated from the posterior-superior bony canal wall in its line of cleavage and the bony wall removed with a narrow rongeur. A narrow Lempert elevator is then inserted between the membranous canal wall and the remaining bony bridge. This elevator must pass into the middle ear parallel to the short crus of the

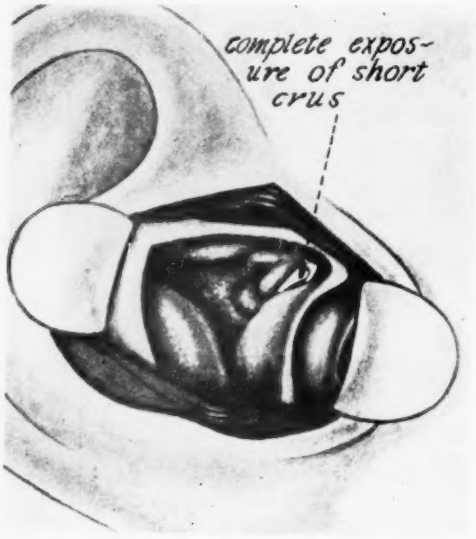

Fig. 6.—Both sides of short crus should be noted before the posterior bony canal wall is removed. (In cases in which there is a narrow angle this cannot always be accomplished.)

incus and must hug the bone in order to avoid tearing the thin membranous ear canal wall. A Lempert rongeur is then inserted over the thinned bony bridge, the superior blade being allowed to rest gently on the short crus of the incus. This maneuver assures the proper depth of the blades before the bony bridge is bitten through. After removal of the bony bridge and the protruding annular ring margins, the epitympanum and its contents are fully exposed to direct view.

A bloodless field is created by repeated use of epinephrine packs of 1 to 1,000 dilution. With magnification, all pathologic tissue, whether

granulation or cholesteatoma, is carefully removed from about the incudomalleal joint area, a small eye spud coupled with gentle suction being used. Some pathologic tissue can also be removed from the upper portion of the middle ear, including the stapes and the stapedius tendon area, without interrupting the continuity of the ossicular chain and its supporting ligaments. If necrosis is found in the ossicular chain so that its continuity has been disturbed, then the incus and the head and neck of the malleus are removed.

Fig. 7.—This exposure completes the modified radical mastoidectomy when the membranous ear canal is left intact: (*1*) superior canal; (*2*) horizontal canal; (*3*) incus; (*4*) malleus; (*5*) suspensory ligament; (*6*) membranous ear canal wall; (*7*) facial ridge; (*8*) chorda tympany nerve; (*9*) stapedius tendon; (*10*) fallopian canal.

In the customary modified radical operation a flap is created from the posterior-superior membranous canal wall and placed over the epitympanum much the same as in the fenestration operation. If the epitympanic pathologic tissue has been satisfactorily removed, I do not create a skin flap but prefer to allow the cavity to heal as in a simple mastoidectomy. In this case the wound is closed by suturing the skin edges of the no. 2 and the edges of the no. 1 and no. 3 incisions together

and placing a small rubber drain in the cavity. By this method, creation of a mastoid bowl or cavity, with its associated healing requirements, is eliminated. In my experience the attic perforation present usually closes spontaneously.

RADICAL MASTOIDECTOMY

If the membranous ear canal wall has been left intact, the radical mastoidectomy is simply a continuation of the completed modified radical operation. Any remnants of incus or malleus, as visualized in the epitympanum, are grasped and removed. The skin flap is prepared by separating the skin of the ear canal from the anterior-superior bony canal

Fig. 8.—Note the skin flap extending posteriorly over the facial ridge.

wall, allowing it to extend posteriorly over the facial ridge to epithelize the mastoid cavity. If one prefers, the entire skin of the ear canal can be excised completely over the facial ridge and discarded or preserved for later use as a free graft. In the event the skin flap is completely discarded, the mastoid cavity heals by fibrosis and the end result is an obliteration of the mastoid cavity and of most of the external ear canal. This procedure eliminates the mastoid bowl, with its problem of epithelization, but should not be used unless all pathologic tissue has been removed from the middle ear area. A skin flap should always be prepared, if one has reason to believe that all pathologic tissue within the middle ear cannot be removed or if the hearing in the opposite

ear is impaired. In the latter instance, a Pohlman[2] artificial ear may be used in an attempt to restore serviceable hearing in the ear operated on.

When the skin flap is preserved, an extremely thin, full thickness elliptical skin graft measuring 1.5 by 3 cm., taken from the postauricular area, has proved of value in decreasing postoperative drainage from the ear. Multiple punctures are made through the graft for drainage of blood serum, and the graft is divided into three portions. On completion of the surgical procedure, one piece is placed over the promontory and eustachian orifice and another over the epitympanum and dural plate

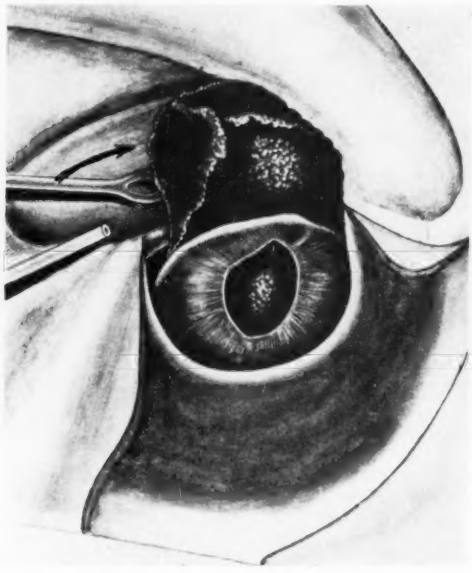

Fig. 9.—The thickened periosteum with the overlying contents of the epitympanum is elevated.

region, and the third covers the sinodural angle. These pieces of skin are held in position by small balls of parresined gauze, which are allowed to remain in place for five days.

Identificaion of the Fallopian Canal.—Before entering the middle ear area it is imperative to visualize the fallopian canal. Most injuries to the facial nerve occur in the fallopian canal area and usually are the result of inadequate visualization of this canal. In the teaching of radical

2. Pohlman, M. E.: Artificial Middle Ear, Ann. Otol., Rhin. & Laryng. **56**:647-657 (Sept.) 1947.

mastoid surgery emphasis has too often been placed on avoiding the area of the fallopian canal in an attempt to prevent injury to the facial nerve. I feel that this teaching is wrong because it not only leads to more injuries to this nerve, due to lack of visualization, but also encourages the operator to leave considerable pathologic tissue about this area, which may result in continued drainage after surgical intervention. Therefore, a technic has been developed to allow early visualization of this canal before any pathologic tissue is removed from the middle ear area.

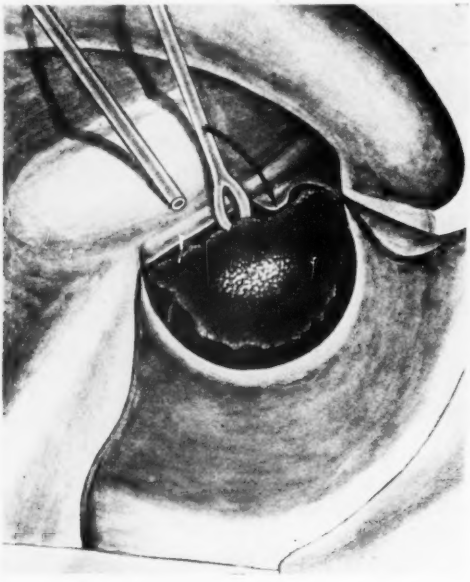

Fig. 10.—The periostum is elevated over the cochleariform process, exposing the fallopian canal to direct view.

After removal of the ossicular remnants and preparation of the skin flap, the posterior portion of the horizontal canal is visualized by again making use of the imaginary line drawn posteriorly from the superior margin of the external bony ear canal. If the ear has been operated on before, the facial ridge, consisting of the remaining part of the posterior bony ear canal wall, is identified and followed to its termination at the drum margin. The horizontal canal will be visualized just posterior to this area.

The thickened periosteum with all the overlying pathologic tissue is elevated in a posterior to anterior direction following the contour of the bony horizontal canal. This maneuver is best accomplished by the use of a small dull ring curet coupled with gentle suction.

This elevation is carried to the far anterior extremity of the horizontal canal, and then the ring curet is directed inferiorly over the cochleariform process into the middle ear. If one enters the middle ear before reaching the far anterior extremity of the horizontal canal and the cochleariform process, dislocation of the stapes may occur. As soon as the cochleariform process is exposed to view, the entire fallopian canal is clearly visualized, even before any surgical procedure is carried out within the middle ear. If this technic is routinely followed, I am confident that

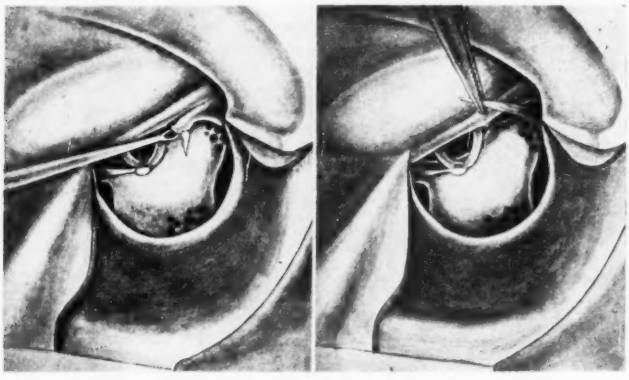

Fig. 11.—Removal of the tensor tympani is routinely performed in radical mastoidectomy.

fewer injuries to the fallopian canal will occur in radical mastoid surgery. "What one sees he will not injure." At this point I must again emphasize the importance of a dry surgical field, adequate illumination and proper magnification.

All pathologic tissue may now be removed from the middle ear area with little fear of injury to the facial nerve. The cochleariform process is fractured by elevating it in an external inferior direction away from the fallopian canal. The tensor tympani is raised from its bony groove by means of a small curet and removed intact.

The inferior margin of the annular ring is removed in order to expose adequately and allow thorough curettement of the hypotympanic area. The eustachian tube orifice is curetted with a no. 0000 Lempert curet, and cotton oxycel is placed in the tubal orifice to add support

to the skin graft.[3] The patient is urgently requested not to blow his nose for a period of two months after surgical intervention, in an attempt to allow complete epithelization to occur over the orifice of the tube.

Removal of Granulations from the Stapes Area.—An ear may continue to drain after a radical mastoid surgical procedure because of infected granulation tissue remaining in the area of the stapes. I have found that these granulations can be removed with little risk of stapes dislocation if one uses a small eye spud and gently sweeps in a posterior

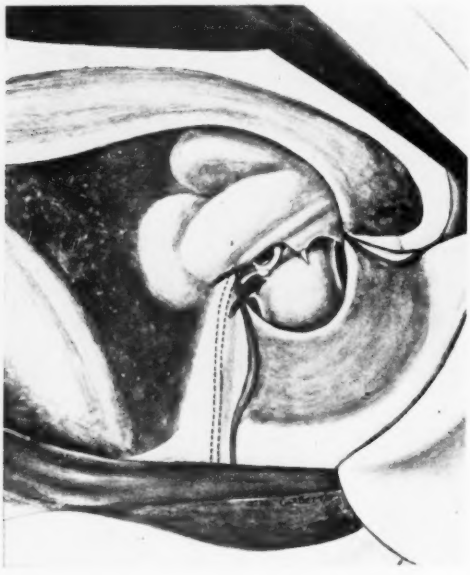

Fig. 12.—Note direction of movement carried out to remove granulations from the stapes area.

to anterior direction above and below the stapedius tendon and stapes. In this way the counterpull of the stapedius tendon acts to prevent tilting of the stapes, whereas an anterior to posterior movement would immediately tilt the stapes from the oval window.

When continuity of the ossicular chain is interrupted, the stapedius tendon may, because of its counterpull, tend to tilt the stapes slightly in the oval window. It has recently been pointed out that this may

3. Walsh, T.: Personal communication to the author.

interfere with the phase differential between the oval and round windows and thereby interfere with hearing.[4] It may be that hearing can be improved after radical mastoidectomy by routinely severing the stapedius tendon.

SUMMARY

A few points in the technic of endaural bone surgery which have proved beneficial in operating for the chronically discharging ear are described.

1. A slight modification of the Lempert incision provides additional skin for later use in lining the mastoid cavity. This same incision is used in all procedures on the temporal bone.

2. Excision of a triangular piece of fascia extending from the posterior membranous ear canal wall to the temporal muscle provides additional exposure and eliminates the necessity of spreading the incision postoperatively in order to prevent atresia of the ear canal.

3. An imaginary line drawn directly posterior from the superior margin of the external bony ear canal will bisect the horizontal canal deeper in the temporal bone.

4. Elimination of the cloth sleeve over the dental arm by sterilization of the arm, belt and engine pulley has proved expedient.

5. Identification of the constant firm bony shelf forming the external wall of the epitympanum has definite value.

6. Except in occasional narrow angle cases, the entire malleus head and neck, together with the short crus of the incus, should be completely visualized before an attempt is made to remove the posterior-superior bony canal wall. This exposure indicates that the bony ear canal wall has been adequately thinned.

7. The narrow Lempert elevator must be parallel to the short crus of the incus as it enters the middle ear if the membranous ear canal tissue is to be separated from the bony bridge without injury.

8. It is not necessary to create a skin flap in the modified radical operation if the pathologic tissue has been satisfactorily removed from the epitympanum.

9. A skin flap may be created or completely removed in the performance of radical mastoidectomy. A thinned, full thickness skin graft may be readily obtained from the postauricular area and, when used, seems to expedite healing of the cavity.

10. Complete visualization of the fallopian canal early in the operation is a must if injury to the facial nerves is to be avoided.

11. Granulations over the stapes area can be more safely removed if one uses a described posterior to anterior maneuver, which takes advantage of the counterpull of the stapedius tendon.

4. Bateman, G. H.: Personal communication to the author.

12. Severance of the stapedius tendon routinely in radical mastoidectomy may result in improved hearing.

CONCLUSION

An intimate knowedge of the surgical anatomy of the temporal bone is an absolute prerequisite in the performance of this operation of chronic otitis. Adequate illumination, proper magnification and the maintenance of a dry surgical field are of paramount importance if the desired results are to be obtained.

1136 West Sixth Street.

ABSTRACT OF DISCUSSION

DR. JOHN F. TOLAN, Seattle, Washington: Dr. House is to be commended on this outstanding presentation in which the technical difficulties encountered are indeed great. Because of widespread interest concerning surgery for the otosclerotic patient, endaural surgery was successfully launched about ten years ago. Lempert did much of the original work in this particular field, followed by Shambaugh, Meltzer, House, Walsh, Maxwell and many others. It has stimulated the otologist to endless research pertaining to the inner ear. Too little investigation regarding the cochlea was done previous to the fenestration operation. We have still not reached the final answer to this problem, but we are progressing along proper lines.

I have a few slides to reemphasize points already brought out by Dr. House in the advantages of the endaural approach. I should like to show the type of skin flap that I am using not only in endaural surgery for fenestrations but also for radical mastoidectomies. In some of my fenestrations and radical mastoidectomies I have encountered persistent postoperative discharge, and I believe this procedure has gone a long way in overcoming this difficulty. The first incision is begun in the membranous lining of the superior-posterior wall of the external auditory canal and the junction of its osseous membranous portions. It is carried straight downward parallel to the osseous membranous junction including one half of the circumference of the canal. The second incision is the same as Lempert's incision. It starts at the point of the first incision in the superior-posterior wall of the canal and is carried outward through the skin of the outer one half of the superior-posterior wall and is continued upward adjacent to the tragus into and along the anterior wall of the suprameatal triangle. The third incision starts at the lower edge of the first incision and runs externally along the floor of the canal through the lower end of the anterior border of the concha. This skin flap is then carefully separated from the under subcutaneous tissue and reflected posteriorly to and exposing the edge of the conchal cartilage. The subcutaneous tissue and the periosteum are then removed, exposing the mastoid cortex. An elliptic piece of the superior edge of the conchal cartilage is removed to create a larger endaural orifice. This skin flap later is folded over the edge of the exposed conchal cartilage. This procedure, I believe, reduces the instances of chondritis and shortens the postoperative care.

We are all familiar with the fact that the endaural approach is the most direct route to the mastoid antrum and middle ear structures, particularly the eustachian orifice. Bleeding is not a troublesome feature with this approach as it is with the postauricular procedure. The type of anesthesia depends on the patient,

varying from local anesthesia with nitrous oxide for some to oxygen and pentothal sodium® given intratracheally.

Dr. House has emphasized that this approach affords a better opportunity of cleaning out all diseased tissues in the mastoid antrum, epitympanum and perieustachian region. It is important that all vestiges of cholesteatomas and granulation tissue be removed. With the aid of the loop or Dr. Shambaugh's microscope, this can carefully be carried out. Trauma to the soft tissues with this approach is at a minimum. Postoperative dressings are not painful. We do not have the ugly postauricular scarring defects and fistulas. The patient is out of bed on the second day and usually is out of the hospital between the third and fifth days. Economically, this is important to the patient, and the postoperative complications are greatly reduced.

Dr. James E. Croushore, Detroit: The development of endaural surgery not only has been necessary for a practical technic in the fenestration operation but also has brought forth a refinement in temporal bone surgery for handling infection. I know several men who took a course in fenestration surgery and then went back to their practice but never performed a fenestration operation. Yet they consider the training most valuable because they learned the surgical anatomy of the temporal bone far better than they thought possible. They became adept at handling an electrically driven burr, they saw the importance of proper illumination and, above all, they discovered that by using magnification a new world in the temporal bone opened to them. Dr. House's presentation has stressed these statements.

The use of the single incision preserves skin and gives a longer flap. If desired, the end of the flap may be amputated, and this piece of skin can be placed in the mastoid cavity as a graft. Excising fascia at the superoanterior aspect of the incision for better exposure and prevention of future atresia of the canal seems like a good procedure, which I will try in the future. Utilization of an imaginary line drawn directly posterior from the superior margin of the external bony ear canal to localize the horizontal semicircular canal deeper in the mastoid and thus aid in early finding of the antrum and aditus is a good idea in helping to keep oriented. The dental arm sleeve is always a nuisance and is a time killer because it gets caught between the pulleys and belt and it is difficult to manipulate the screw at the end of the handpiece through it. Elimination of the sleeve by autoclaving the dental arm together with the belt and engine pulley certainly would be a mechanical improvement.

Dr. House made a good point when he stressed the outlining of the bony shelf which forms the external wall of the epitympanum. This bony landmark represents a definite "stop sign" in the complete or cortical mastoidectomy and a "go sign" in a more radical mastoidectomy.

If complete visualization of the fallopian canal is attained in every radical mastoidectomy, as Dr. House suggested, there certainly would be fewer facial nerves injured. The destructive processes and the great amount of disease present in some ears may not permit such an orderly procedure as one would like. Considerable debris may have to be removed before any semblance of a middle ear is apparent. In these instances extreme caution is required so as not to injure the nerve, stapes or even the round window.

Dr. House does not turn a skin flap in some modified radical mastoidectomies. I have not employed this procedure, but I believe it merits trial in carefully chosen cases. The decision can be made only during the operation.

In the modified radical mastoidectomy extreme care must be taken to avoid dislocating the incus. If good hearing is present—and this is one of the indications

for the modified procedure—the incus must maintain its normal position. Rarely will the hearing maintain the preoperative level once the incus is dislocated.

There are several ways of handling the skin flap in the radical operation. I have always utilized it in some manner and never discarded it. On several occasions I have cut the flap in its center in its long axis and turned the one half upward and the other down over the facial ridge.

I have had no experience with the Pohlman artificial ear. Properly employed, it should be effective in improving the hearing in ideal cases.

Dr. O. Popper, Johannesburg, South Africa: I should like to add my sincere admiration for the presentation of this paper. There is not the slightest doubt in my mind that the only reasonable operation to be done on a chronically infected mastoid is by the endaural route. But there is another group of cases in which the mastoid is sclerotic and in which you might think it is advisable not to disturb what, after all, is nature's method to seal off the infection.

I had the opportunity this morning of demonstrating a direct approach to the attic and antrum by the transtympanic route. When the mastoid shows signs of infection roentgenologically, obviously you must clear it out by the endaural route. But when you are reasonably sure that the mastoid is sclerotic and, as I say, healed, I should like to urge you to try the procedure of clearing only the atticoantral region by the transtympanic route. I hope that some who watched me doing it this morning were convinced. I have used this method in 27 cases, in each of which I have made sure that the mastoid is sclerotic; the results in this small series have been encouraging—rapid epithelization, shortened postoperative period of otorrhea and preservation of hearing. We have come to expect destruction of hearing in the radical operation!

The transtympanic approach is not a difficult operation and certainly less traumatic; but the main point about it is that if the hypotympanum is cleared at the same time and if the hypotympanum (that is, the region in the vicinity of the round window) can be kept clear, one will restore hearing as well as control discharge.

The point which I am trying to make is whether we could not shift the center of gravity in our operative outlook and decide that the first consideration in operating on any chronically diseased ear shall be that we shall restore hearing and the second that we shall control discharge. Naturally, when the mastoid is infected one has no option but to do the classic endaural operation, but if operation on the mastoid can be avoided, one may, in addition to controlling the discharge, restore hearing.

Dr. Howard P. House: I agree with Dr. Tolan that preservation of skin for the purpose of lining the cavity may help prevent annoying postoperative drainage due to incomplete epithelization. Creation of a small cavity has definite value in the fenestration operation.

I agree with Dr. Popper that no "man-made job" is as good as nature's. Certainly, unnecessary removal of noninfected bone is to be avoided, whenever possible.

MÉNIÈRE'S SYNDROME

Observations on Vitamin Deficiency as the Causative Factor

I. The Vestibular Disturbance

MILES ATKINSON, M.D., F.R.C.S. (Eng.)
NEW YORK

MÉNIÈRE'S syndrome may be regarded as consisting of two phases: (1) the acute, the attacks of vertigo or the vestibular disturbance; (2) the chronic, the deafness and tinnitus or the cochlear disturbance, which persists between the acute paroxysms. The present paper is concerned solely with the acute phase. The chronic phase will be dealt with in a subsequent communication.

In earlier papers [1a, b] it was reported that patients with Ménière's syndrome could be divided into two groups by determining the degree of response to an intradermal injection of histamine performed under standard conditions.[2] Clinical experiments were described which showed that in the group which gives a small or negative response the symptoms are improved by vasodilator drugs and made worse by vasoconstrictor drugs, while in the group which gives a large or positive response the reverse is the case. These findings were interpreted as evidence for a vascular mechanism as the basis of the characteristic recurring attacks of vertigo—in the one group (the histamine-negative) a primary vasospasm, and in the other (the histamine-positive) a primary vasodilation—and treatment directed toward overcoming the particular vascular disturbance in each group has proved reasonably satisfactory. These observations led inevitably to the question—what is the factor, or what are the factors which produce these disturbances of vascular function? The purpose of this paper is to put forward clinical evidence in support of the thesis that one such factor, perhaps the essential factor, is a nutritional, more specifically, a vitamin, deficiency.

Read before the Central New York Eye, Ear, Nose and Throat Society, Rochester, N. Y., April 28, 1948.

1. (*a*) Atkinson, M.: Observations on the Etiology and Treatment of Ménière's Syndrome, J. A. M. A. **116**:1753 (April 19) 1941; (*b*) Histamine in the Treatment of Ménière's Syndrome: An Appraisal, ibid. **119**:4 (May 2) 1942; (*c*) Ménière's Syndrome: Results of Treatment with Nicotinic Acid in the Vasoconstrictor Group, Arch. Otolaryng. **40**:101 (Aug.) 1944; (*d*) Ménière's Syndrome: Its Mechanism and Management, New York State J. Med. **44**:489 (March 1) 1944.

2. To insure accuracy in judging this test, it is important that there be transverse lighting with a flashlight in a darkened room. In this way a pseudopod will often be seen which with direct lighting is not visible.

PRELIMINARY OBSERVATIONS

It will make for better understanding of the statistical tables and of the method of treatment to be described later if first a brief account is given of how they came to be arrived at.

Since the original papers on the use of nicotinic acid in the treatment of patients with Ménières' syndrome were published,[2a] this substance has come to be widely used, and satisfactory results obtained with it in a large series of cases of the vasoconstrictor group have been reported.[1c] In the past it has been the contention that in this group the effect of nicotinic acid depended solely on the vasodilator action, but the possibility that it was due to a vitamin action has always been present. It will be seen later that in these cases clinical signs of niacin deficiency are actually present.[2b]

Although the results of nicotinic acid therapy in the vasoconstrictor group were in general satisfactory as regards relief of the major attacks of acute rotational vertigo with vomiting, many of these patients continued to experience positional vertigo, manifested by momentary flashes of "dizziness," particularly on sudden movements of the head, or even by longer-lived periods of general unsteadiness. In a few cases, after an initial satisfactory response, attacks seemed to be induced by the continued exhibition of nicotinic acid, and once or twice in the most severe cases an attack undoubtedly has been produced by intravenous injection. The type of attack, however, has been different, and from being originally of the rotational type with vomiting it assumed the character of lateral or vertical movement of objects, now usually without vomiting, though with nausea. In the most severe cases the

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2a. Harris, H. E., and Moore, P. M., Jr.: The Use of Nicotinic Acid and Thiamin Chloride in the Treatment of Ménière's Syndrome, M. Clin. North America **24**:533 (March) 1940. Atkinson.[1a]

2b. The word "niacin" is used here as a general term to denote the nicotinic radical. The word was compounded originally from three words, *nic*otinic *a*cid vitam*in*, and therefore, strictly speaking, should be used only as an alternative for nicotinic acid. Since the advent of nicotinic amide, however, niacin has come to be used as synonomous with both the amide and the acid. This has led to so much confusion that even the term "niacinamide" has been coined. Nutritionists now seldom use nicotinic acid for prevention or treatment of deficiency, preferring nicotinamide because it does not produce what is regarded as the undesirable vasodilator effect associated with nicotinic acid. However, in the treatment of patients with certain clinical conditions, of whom the vasoconstrictor group of patients with Ménière's syndrome is an example, the acid is preferable to the amide precisely because of this effect. As a way out of this difficulty of terminology, the term "niacin" has been used in this article in a generic sense to include both substances; and when one or other of the nicotinic derivatives is specifically intended, it has been referred to as nicotinic acid or nicotinamide. From the clinical, as opposed to the nutritional, point of view the two are not interchangeable (see page 171).

effect was that of a sudden knock-down blow. At the same time as this change in clinical type took place, the results of the histamine skin test changed from negative to positive. Moreover, now that awareness of possible vitamin deficiency had come to consciousness it was observed that these patients presented the signs of riboflavin deficiency in addition to those of niacin deficiency. Riboflavin was therefore administered, and it was found that by this means the new attacks could be controlled.

In addition to the attacks of vertigo many of these patients complained of great fatigue and "weak spells." These had often been present from the beginning in some degree but became more pronounced as nicotinic acid and riboflavin continued to be given, and in addition there arose nervousness, jitteriness and irritability. When thiamine was thereupon added, these symptoms also subsided.

In this way it was found possible by administration of the appropriate vitamin to remove symptoms layer by layer as on a palimpsest. As one layer was removed so, the next became evident. Cases 1 and 2 were the first of several in which the patient was so observed and treated.

CASE 1.—The patient, a woman aged 31 years, was first seen in January 1943. She had a typical Ménière's syndrome with associated headache in the frontal and occipital areas on the affected side. The attacks of vertigo had started one year previously; they occurred at first about once a month in the winter only and were of two types. In one type of attack she experienced severe rotational vertigo with vomiting. In the other type the sensation was of lateral movement of objects without vomiting but with severe nausea. In addition, she complained of oozing from both ears, particularly at the time of an attack (this was otitis externa) and of considerable fatigue. The result of the histamine skin test being negative, she was treated with nicotinic acid, which gradually controlled the acute attacks of rotational vertigo with vomiting, but had no effect on the less severe attacks in which objects moved from side to side.

A year later, in January and February 1944, she had three attacks of positional vertigo in six weeks, none of which she called severe, because they were short-lived and she did not vomit, though in the last of these she had for the first time fallen to the ground. The ears had been oozing badly for six days previously. During this six weeks her dose of nicotinic acid had been increased because of the attacks, her husband, a doctor, giving her daily injections in addition to her oral medication. These injections seemed to have increased the frequency of the attacks. In consequence, they were stopped, but the oral dosage was continued at a reduced level. In April 1944 she had one or two mild attacks, but then the attacks ceased and on the lower oral dosage of nicotinic acid she was well until September 1945, when she again had two knock-down attacks and was nervous. She did not return, however, until April 1946, after having had, in the interval, one mild rotatory attack and two more knock-down attacks, the second two days before in a restaurant, where she fell face forward into her soup. Her ears were oozing profusely. At this time a histamine skin test gave a small positive reaction. During all this time she had been taking nicotinic acid in varying dosage, but consistently. To this in

the light of the new concept was now added riboflavin. Three weeks later she reported that she had had one mild attack only and that the ears had stopped oozing.

In October 1946 she reported that she had had no further attacks of either type but that she was extremely nervous and upset, indeed panicky, and complained of fear of heights, of crowds, of the subway and also of extreme fatigue. She was thereupon given thiamine hydrochloride by injection and by mouth in addition to the nicotinic acid and the riboflavin. A month later, in mid-November 1946, she said that she felt "like her old self again for the first time in five years." Since then until last seen in mid-November 1947 she has been treated constantly with oral dosage of nicotinamide, riboflavin and thiamine, has had no attacks, no headache, no undue nervousness, has been through a pregnancy sucessfully without recurrence of symptoms, and her otitis externa has resolved.

CASE 2.—This is another similar case. The patient was a woman, aged 49 years. Nicotinic acid, after at first relieving major attacks of rotational vertigo, which were occurring two or three times a month, later was found to induce attacks, though now they were of a positional type. At the same time the patient became extremely irritable, was at times quite rude when making appointments over the telephone, and on one occasion, after having been kept waiting five minutes before being seen and having paced the waiting room the while, refused to wait any longer and walked out. When later she returned, she was given riboflavin in addition to nicotinic acid, which rapidly controlled the new attacks, and soon thereafter thiamine hydrochloride, a procedure which rapidly turned her from a difficult, uncooperative patient into a pleasant, placid one who returns regularly for a routine check-up. She has now been free from attacks of any sort for twelve months.

Thus it began to appear that the two main types of vertigo of which these patients complain, rotational vertigo, on the one hand, and forced movement or positional vertigo, on the other, are in fact two separate entities; that each is associated with a specific deficiency which can be correlated with the result of the histamine skin test; that, moreover, many patients suffer from both types of vertigo and from both deficiencies.

The obvious next step was to investigate the histamine-positive group from the standpoint of vitamin deficiency, and, as will be seen later, what was to be expected from the findings in the cases described was observed, that in this group the predominant deficiency is of riboflavin and the type of vertigo is positional.

In the light of these findings it became necessary to revise the method of grouping patients according to their response to the histamine skin test. Whereas previously they had been divided into two groups only, those with negative and those with positive results, it now became necessary to introduce a third or intermediate group consisting of those who showed a large response without true pseudopodia (large negative reaction) and those who showed one or more small pseudopodia (small positive reaction) instead of the one or more long, trailing pseudopodia which constitute the true positive response. The remainder of this

paper will describe the observations made in a series of cases of typical Ménière's syndrome from the new point of view just outlined.

MATERIAL AND PROCEDURE

Material.—Eighty-seven consecutive cases of the classic Ménière's syndrome, i. e., cases showing the full-fledged triad of acute paroxysmal vertigo, deafness and tinnitus, predominantly or entirely unilateral, have been investigated from the standpoint of deficiency in the three major components of the B complex (niacin, riboflavin and thiamine) during the twenty-one months from March 1946 through November 1947.

Procedure.—The procedure for each patient has been:

1. To record the response to a standard dose of histamine diphosphate administered intradermally under standard conditions as described elsewhere.[1d]

2. To note the types of vertigo complained of by each patient.

3. To keep a chart of those symptoms generally accepted as accompanying certain vitamin deficiencies (chart).

VITAMIN A	RIBOFLAVIN—*Continued.*
Night blindness	Corneal vascularization
Hyperkeratosis	Pinguecula
Dental caries	
THIAMINE	NIACIN
Anorexia	Tongue
Constipation	Abdominal tenderness
Loss of weight	Anorexia
Paresthesias	Diarrhea
Muscular tenderness	Dermatitis
Fatigue	Mental depression
Loss of tendon reflexes	Mental confusion
Palpitations	Loss of memory
Tachycardia	Headache
Cardiac enlargement	Vertigo
	"Feel the cold"
RIBOFLAVIN	ASCORBIC ACID
Cheilosis	Gums
Seborrheic dermatitis	Joints
Magenta tongue	Hemorrhages
Photophobia	Weakness
Lacrimation	
Eye strain	

Case sheet for nutritional deficiencies.

4. To make a corresponding physical examination, noting signs of niacin deficiency in the tongue; of riboflavin deficiency in the eyes, the lips, the skin of the face and in the tongue; of thiamine deficiency in disturbances of function of the nervous system as shown by hypereflexia or hyporeflexia.

5. To record by means of color photographs the appearances of the organs mentioned in the foregoing paragraphs.

6. To treat each patient with the particular fraction appropriate to his group and with that alone in the first place and to observe and record the results of such treatment.

RESULTS OF EXAMINATION

Using as a yardstick for niacin deficiency the criteria laid down by Kruse for the tongue,[3] for riboflavin deficiency the criteria of Kruse and co-workers[4] for the tongue and eyes, and for thiamine deficiency the degree of activity of the patellar tendon reflex, and associating these objective findings with the subjective complaints, an assessment was made in each case of the degree of deficiency of each of what Spies has called the big three of the B complex. Four degrees of deficiency were used in the assessment—1 and 2 plus representing degrees of mild chronic deficiency, and 3 and 4 plus, degrees of severe chronic deficiency.

By these criteria every patient showed evidence of chronic deficiency of these three factors in greater or less degree, while in some, in addition, acute manifestations would be superimposed on the chronic background at a time of attacks and be manifested by such complaints as sore tongue, diarrhea, angular stomatitis, sore eyes, oozing ears (case 1), fatigue, tachycardia and palpitations. Although in many cases deficiency of one factor predominated, evidence of some deficiency of the other two was almost always present, and in some there was more or less equal deficiency of all three (tables 1, 2 and 3). This was to be expected if deficiencies were found at all, since vitamin deficiencies like other troubles seldom come singly.

Further, it was found that the results of the histamine skin test could be correlated with the predominant deficiency.

Group 1. Patients Whose Reactions to Histamine Were Negative.—The 20 patients of this group—23 per cent of the total number of patients—showed predominant niacin deficiency of the chronic type (table 1). In 16 of the 20 patients the deficiency was severe, while in only 4 was it mild. As against this, riboflavin deficiency in this group was predominantly mild; in 15 of the 20 patients it was of

3. Kruse, H. D.: The Lingual Manifestations of Aniacinosis, with Especial Consideration of the Detection of Early Changes by Biomicroscopy, Milbank Mem. Fund Quart. **20**:262 (July) 1942.

4. Kruse, H. D.; Sydenstricker, V. P.; Sebrell, W. H., and Cleckley, H. M.: Ocular Manifestations of Ariboflavinosis, Pub. Health Rep. **55**:157 (Jan. 26) 1940.

this degree, and in only 5 was it severe and in none of them was it classed as 4 plus. Thiamine deficiency also was mainly mild, 11 of the 13 patients for whom this deficiency was recorded having the mild grade and only 2 the 3 plus grade and none the 4 plus grade.

TABLE 1.—*Deficiencies in the Histamine-Negative Group (20 Cases)*

Degree of Deficiency		Cases in Which Given Degree Was Noted					
		Nicotinic Acid		Riboflavin		Thiamine	
0		0		0		0	
+	Mild	0	4	2	15	6	11
++		4		13		5	
+++	Severe	13	16	5	5	2	2
++++		3		0		0	
Total			20		20		13 (7 unrecorded)

TABLE 2.—*Deficiencies in the Histamine-Positive Group (24 Cases)*

Degree of Deficiency		Cases in Which Given Degree Was Noted					
		Nicotinic Acid		Riboflavin		Thiamine	
0		0		0		1	
+	Mild	8	24	0	7	2	10
++		16		7		8	
+++	Severe	0	0	11	17	10	10
++++		0		6		0	
Total			24		24		21 (3 unrecorded)

TABLE 3.—*Deficiencies in the Intermediate Group (43 Cases)*

Degree of Deficiency		Cases in Which Given Degree Was Noted					
		Nicotinic Acid		Riboflavin		Thiamine	
0		0		0		3	
+	Mild	5	18	2	16	10	25
++		13		14		15	
+++	Severe	22	25	21	27	14	14
++++		3		6		0	
Total			43		43		42 (1 unrecorded)

Group 2. Patients Whose Reactions to Histamine Were Positive.—The 24 patients—27 per cent of the total number of patients—showed, by contrast, predominant deficiency of riboflavin (table 2). In 17 of the 24 patients the deficiency was severe; in only 7 was it mild, and in these it was 2 plus. Niacin deficiency, on the other hand, appeared mild in every patient. Thiamine deficiency tended to be more severe in this group; 10 of 21 recorded were classed as having

severe deficiency (3 plus) and 10 as having mild deficiency, while 1 showed no objective evidence of thiamine deficiency.

Group 3. An Intermediate Group, Whose Reactions Were of the Large Negative or Small Positive Type.—These 43 patients—50 per cent of the total number—formed the largest group. In this group deficiencies were fairly evenly divided, though as far as concerns nicotinic acid and riboflavin the majority were of severe degree (table 3). Niacin deficiency appeared mild in 18 of the 43 patients and severe in 25. Riboflavin deficiency was mild in 16, severe in 27. Thiamine deficiency tended to be mild; 4 of the 42 recorded had no evidence of deficiency, 25 had a mild degree, and 14 a severe degree, 3 plus only.

Thus the result of the histamine skin test can be translated into terms of niacin or riboflavin deficiency and can be used, and effectively used, as a check on the physical findings. A negative result is associated with a predominating niacin deficiency, a positive result with a predominating riboflavin deficiency, and an intermediate response with fairly evenly mixed deficiencies of both fractions.

TABLE 4.—*Types of Vertigo in Groups*

Group	Rotational	Mixed	Positional	Total
Vasoconstrictor (histamine −).....	*5	14	1	20
Intermediate (histamine ±).....	3	34	6	43
Vasodilator (histamine +).....	2	8	14	24
Total.....	10	56	21	87

TYPES OF VERTIGO RELATED TO SPECIFIC DEFICIENCIES

The fact that many patients complain of two different types of vertigo, rotational and positional, that change of predominance of one type over the other may take place under treatment and be accompanied by change of response to histamine intradermally injected, and that it has been possible to control one type with one vitamin and the other with another (cases 1 and 2) suggested, as has been said, that a specific type of vertigo is associated with a specific deficiency. The findings in the present series bear out this assumption (table 4).

In the histamine-negative or predominantly niacin-deficient group 5 of 20 patients described the vertigo as rotational only, 1 as positional only (in this case lateral movement of objects), the remaining 14 experiencing both rotational and positional vertigo. The bare figures, however, do not give the whole story. In all 14 of those experiencing both kinds of attack, the severe attacks were associated with rotational vertigo, while positional vertigo consisted only of momentary "flashes" of imbalance or some unsteadiness between attacks. Thus, in 19 of the

20 patients of the niacin-deficient group the predominant or sole type of vertigo was rotational.

By contrast, in the histamine-positive or predominantly riboflavin-deficient group 14 of the 24 patients experienced vertigo of positional type only, 2 described rotational vertigo only, and 8 described attacks of both types with positional vertigo the more severe. Here again the bare figures do not delineate the picture accurately. Positional vertigo was always much more severe than in the previous group, attacks were longer lived, often disabling, associated with severe nausea, though seldom with vomiting, while several patients experienced the most severe degree of positional vertigo, a knock-down blow, even occasionally with momentary unconsciousness. Thus, in this group with a predominating riboflavin deficiency positional vertigo was the sole or the more severe type in 22 of the 24 patients.

In the intermediate or mixed deficiency group vertigo of both types and of varying degrees of severity occurred in 34 of the 43 patients, with only 3 describing rotational vertigo alone, only 6 positional vertigo alone.

It appears from these observations that the type of attack, the result of the histamine skin test and the signs of specific deficiencies can all be tied in together. It is a fascinating exercise to predict from the clinical history the result of the histamine test and the predominating deficiency, and it can be done, after a little practice, wth surprising accuracy.

THE THERAPEUTIC RESPONSE

The final step was to put the observations recorded in the foregoing sections to the test of therapeutic response by treating members of each group with the appropriate vitamin fraction and so far as possible with that alone, at least until satisfactory evidence of its effect had been obtained. The criterion of effectiveness has been control of attacks. Just as in describing the clinical picture, consideration has been directed entirely toward the type of vertigo experienced, and all mention of the cochlear disturbance deliberately omitted, so control of vertigo has been used as the test of therapeutic response. As was said at the beginning, the acute phase alone is dealt with in this paper, without regard to the chronic background. The cochlear disturbance will be considered in a later communication.

Group 1. Niacin Deficiency and Negative Reaction to Histamine.—Observations on 17 of the 20 patients of this group are available for report (table 5). All these patients were treated initially with nicotinic acid alone. In every one the attacks were rapidly brought under control, and though, as often happens, minor relapses occurred from time to time, these became increasingly less severe and less frequent until in a few months complete control, even of minor manifestations, had been established. All 17 patients have been entirely relieved of vertigo

of any degree for periods of six months to four years (5 had been under treatment with nicotinic acid before this investigation started, and the observations were included in a previous report [1c]).

The method of treatment was that already described: intravenous and intramuscular injection, and coincident oral administration which was continued as maintenance treatment for an indefinite period. The only difference in this series has been that in the early stages doses tended to be higher, whereby more speedy control was obtained, and that, once control had been established, a mixed vitamin capsule was added to maintain vitamin balance.

These results are better than those reported previously in a much larger series.[1c] In that series a number of patients who would now, in the light of the present investigation, be placed in the intermediate group, were classified then as histamine-negative and treated accordingly. A high proportion of successes as regards relief of major rotational attacks

TABLE 5.—*Results of Treatment with Reference to Control of Attacks of Vertigo*

		Controlled	Improved	Unchanged	Total
Vasoconstrictor group					
Cases—17		17	0	0	17
Deficiency—nicotinic acid					
Intermediate group					
Cases—38					
Deficiency—combined	nicotinic acid	V_R 31	5	0	36 (see text)
	riboflavin	V_P 23	14	1	38
Vasodilator group					
Cases—18		7	11	0	18
Deficiency—riboflavin					

"Controlled" signifies that the patient has been completely relieved of vertigo but is still under maintenance treatment.
"Improved" signifies that the patient has been freed from major attacks but still has occasional minor experiences of vertigo or unsteadiness.
V_R means rotational vertigo.
V_P means positional vertigo.

was obtained, but many had minor residual manifestations of occasional short-lived positional vertigo which they discounted.

The essential point that has been brought out is that the rotational vertigo characteristic of this group can be completely controlled with nicotinic acid alone, that it is, in fact, a manifestation of niacin deficiency.

CASE 3.—A woman aged 23 years, first seen on April 25, 1946, had had five attacks of rotational vertigo with vomiting in the previous five years, the last two on April 3 and 5, 1946. Deafness and tinnitus, present at first only at the time of attacks, had been constant since the third attack which occurred in July 1945. She presented marked signs of niacin deficiency in the tongue and gave a true negative response to the histamine skin test. She was given nicotinic acid at first intravenously for two weeks, the daily dose being gradually increased to 50 mg., during which time she had two mild attacks without vomiting. She was then given intramuscular injections, 75 mg. being administered daily for another month. During this time she took 150 mg. daily by mouth, a dosage which was continued after the intramuscular injections were stopped.

A year later she had had no further attacks. At this time she was told to take a mixed vitamin capsule daily and has not been heard from since.

CASE 4.—A man aged 55 years was first seen in November 1945. He complained that he had undergone attacks of rotational vertigo at intervals of three to four months during the previous five years and that there had been deafness and constant tinnitus in the left ear for eight years. The frequency of the attacks had recently increased, four having occurred in the previous two months. He gave a true negative response to the histamine skin test. He was given nicotinic acid. The method of administration was the same as, and the dosage similar to, that in case 3. After the third intravenous injection, his head felt clearer, and the tinnitus started to diminish. Seen in March 1946, when the present investigation began, he was observed to have signs of niacin deficiency in the tongue and mild signs of riboflavin deficiency. He was given a mixed vitamin capsule in addition to his nicotinic acid tablets, which he had been taking steadily, and he has continued this treatment to the present time. In spite of two attacks of influenza, he has had no vertigo since treatment started, and his head is clear except for a bad day about once in six months. He continues his oral medication religiously.

Group 2. Riboflavin Deficiency and Positive Reaction to Histamine.—Observations on 18 of the 24 patients of this group are available (table 5). Of these, 7 have been completely relieved of their attacks of vertigo, 11 have been improved to the extent that major attacks are under control and minor manifestations, such as occasional periods of unsteadiness or momentary "flashes" of vertigo on sudden movement of the head, are becoming steadily less frequent. The duration of the results extends from six to fifteen months.

It has not been possible to make treatment quite so clearcut in this as in the previous group. In every case riboflavin alone was used initially, and in 9 cases attacks have been controlled completely by its use alone. In the remaining 9, in which it appeared as though control was not being obtained or was inadequate, recourse has been had to the former method of treatment of patients of this group, histamine desensitization,[1d] in addition to the administration of riboflavin, and this proved rapidly effective. The conjunction of the two methods appeared to produce more rapid and more effective control than had been obtained in the past with histamine desensitization alone. The 9 cases in which riboflavin alone failed were all cases encountered in the early stages of this investigation, and failure may well have been due in part to lack of experience and inadequate dosage. As experience has increased and with it intensity of treatment, the results obtained with riboflavin alone have improved and are improving, so that latterly recourse to histamine desensitization has not been found necessary.

With riboflavin a method of treatment has been used similar to that with nicotinic acid. Intravenous injections of riboflavin alone were given initially, in the beginning of the investigation, the dose increasing at first from 3 to 10 mg., given daily or on alternate days, and later from

5 to 15 mg., the injections numbering from 20 to 40, according to the response and control of attacks. At the same time riboflavin has been given by mouth, the dose increasing at first from 20 to 80 mg. a day, later from 40 to 160 mg. and even 200 mg. a day. As in the previous group, when control of attacks seems to have been achieved and the point of the experiment established that such control could be effected by treatment of the preponderating deficiency alone, other vitamins were given as supplementary therapy to preserve vitamin balance.

Of the 9 patients treated with riboflavin alone, 5 have been completely relieved of their attacks of positional vertigo, while the remaining 4 have been relieved of acute attacks and now have only minor attacks, which are becoming progessively less frequent. Of the 9 patients who have had supplementary histamine desensitization, 2 have been completely relieved; the remainder are improved and improving on riboflavin alone, given in increased dose. Though the evidence is not as definite as that for nicotinic acid in the previous group, it suggests rather strongly that the positional vertigo characteristic of this group can be controlled with riboflavin alone; in other words, that positional vertigo is a manifestation of riboflavin deficiency as rotational vertigo is a manifestation of niacin deficiency. This is further borne out by the findings in group 3.

CASE 5.—A man aged 62 years, first seen in July 1947, had started to have attacks of vertigo six months previously. The attacks were of gradually increasing frequency until, when seen, he was having as many as three a week. The vertigo was of a positional type with lateral movement of objects and occasional lateral pulsion. He was nauseated in the attacks and on one or two occasions had vomited. Deafness had been noted in the left ear since the onset of vertigo, with occasional tinnitus. He gave a positive response to the histamine skin test and showed signs in the eyes of severe chronic riboflavin deficiency. He was given 10 mg. of riboflavin intravenously for eighteen injections. At the same time he took by mouth at first 40 mg. of riboflavin a day and later 80 mg. a day. On the fourth day after treatment started, he had a severe attack lasting two hours and felt unsteady for three days afterward. After that he had no more attacks, though on three occasions he felt unsteady and "groggy" for half an hour or so. At the end of a month he said that his head felt clearer than it had for months and that he himself in general felt better. He continues free of attacks while taking riboflavin, 120 mg. daily, plus a mixed vitamin capsule; indeed, he is so well that he cannot be persuaded to return for reexamination.

CASE 6.—The patient, a woman aged 35 years, was first seen in June 1947. She complained that for the past nine years she had suffered from attacks of "dizziness." In these attacks she had continuous unsteadiness for as long as six days at a time with a tendency to walk to the left and with exacerbations, in which any movement of the head would provoke extreme dizziness. During these attacks she was nauseated but never vomited. At first she had free intervals of several months, but by the time she came for consultation the attacks had increased greatly in frequency, so that during the previous six months she had been having one or two a week. These attacks were short lived, lasting half an hour only, but were severe, though without any background of unsteadiness and still without vomiting. She did not suffer from deafness or tinnitus until

January 1947, when she awoke one morning with severe tinnitus of a roaring, booming character and a severe loss of hearing in the left ear, both of which had remained constant since their onset. She had had "sinus" headaches, two or three a year, for the past twenty years. She showed signs of severe chronic riboflavin deficiency, and mild chronic niacin deficiency. She gave a markedly positive response to the histamine skin test.

Treatment was started with riboflavin, given intravenously and by mouth as described in case 5, and except for a mild attack on the fourth day of treatment and an occasional momentary unsteadiness during the next week she had no more positional vertigo. Early in July, however, her left ear began to feel "tight" again, and her tinnitus became slightly louder. As it was thought that this might presage an attack, histamine desensitization was started, and intravenous injection of riboflavin was continued. The desensitization may or may not have been necessary. From that time until the present (five months) she has had no attack of vertigo, only a feeling of fulness in the ear at infrequent intervals. Tinnitus has diminished; deafness remains unchanged. In general, she feels much better, particularly in the past two months since she began taking mixed vitamins in addition to her riboflavin. She was given twenty-two intravenous injections of riboflavin in all, and the dose has been increased to 200 mg. a day, taken by mouth, which is producing continuing improvement.

CASE 7.—A man aged 33 years was first seen in August 1944 complaining of attacks of positional vertigo which had been increasing in frequency for the past five years. When seen he was having one or two a week with frequent falls and once or twice momentary unconsciousness. They would last from five minutes to an hour and were associated with nausea but never with vomiting. He had mild loss of hearing in the right ear with occasional tinnitus. He gave a positive response to the histamine skin test. A course of histamine desensitization promptly relieved the attacks, with the hearing restored to normal and the tinnitus abolished.

In January 1945 he had a relapse, which was relieved by a second course of histamine desensitization. In February 1947 he returned with a second relapse, of a relatively mild nature, and it was then noted that he showed signs of severe chronic riboflavin deficiency. On this occasion, therefore, instead of histamine desensitization, intravenous and oral administration of riboflavin was carried out as described, and he promptly improved. After four intravenous injections of riboflavin, 10 mg. being given in each, his positional vertigo ceased, the sensation of fulness of the ear disappeared, and he said that he felt better than he had for several years. He was given twelve intravenous injections of riboflavin altogether and 80 mg. a day by mouth. By April he was symptom free.

In September 1947 he returned, having had a mild relapse after a period of severe nervous strain due to the illness and ultimate death of his wife. His symptoms were again immediately controlled with riboflavin, 10 mg. being given intravenously, and amounts increasing to 120 mg. orally, each day. Since then he has been well and free from symptoms. He continues taking riboflavin and mixed vitamins.

CASE 8.—As the patient was first seen early in January 1948, this case is not included in the present series. It is reported briefly here because it demonstrates rapid control of severe vertigo with riboflavin only. The patient was a man aged 47 years. From 1942 through December 1947 he had experienced about two severe attacks of vertigo a year, accompanied by nausea only. He staggered in the attacks, walked toward the right and was forced to go to bed because movement of the head made him much worse. Before these attacks

he would have loss of hearing and tinnitus in the right ear and a sense of fulness, all of which were relieved by the attack (Lermoyez type). In December 1947 he had four attacks of the same sort, and from Christmas Day to New Year's Day he had daily attacks. For one year before this the deafness and the tinnitus of the right ear had been increasing and were not always immediately relieved by the attack, and since December 1947 both had been constant.

Previous treatment had been interesting in its effects. Up to December 1947 eustachian catheterization had seemed to be effective—"when the ear was opened vertigo ceased, but now they cannot keep it open for more than a few hours at a time." On Dec. 24, 1947 he was given nicotinic acid orally for four days and had four attacks. (See comment under "Nicotinic Acid vs. Nicotinamide," page 170).

When seen he showed no spontaneous signs of disease of the labyrinth, had a 50 per cent loss of hearing in the right ear and definite signs of riboflavin deficiency. The histamine skin test gave a positive reaction.

Treatment was started with riboflavin, 10 mg. being given intravenously and 40 mg. by mouth daily. After the tenth injection he reported that all vertigo had disappeared, that his ear was clear, that the noises had ceased and that he felt better than he had for months. By that time his daily oral dose had been increased to 80 mg. Then he had a relapse. His daily intravenous dose was stepped up to 20 mg. and his daily oral dose gradually to 200 mg., and control was again obtained. Usually an injection would "open" his ear and thus relieve his vertigo within half an hour, and this relief would be maintained for several hours. One day he came into the office, unsteady and nauseated, an attack having started while he was being driven in. An intravenous injection of 20 mg. of riboflavin had no effect; so a second injection was given twenty minutes later. In fifteen minutes his ear started to "open," and in forty-five minutes he was free from vertigo. Oral dosage was then tried as a means of controlling an attack, and on three occasions he has been able to do this by taking 50 mg. in one dose by mouth. At the time of writing he has had forty-six injections; his threats of attacks are now infrequent and becoming steadily milder, and the trick of control seems to have been learned, at least in this case. He states that he feels better than he has since the attacks first started, and there has been a great change for the better in his appearance in two months of treatment.

Group 3. Mixed Deficiencies and Intermediate Reaction to Histamine.—Observations on 38 of the 43 patients of this group are available for report (table 5). All these patients suffered from vertigo of both types, rotational and positional; 2 had undergone section of the eighth nerve before being seen, and their attacks of rotational vertigo had ceased, but positional vertigo continued after operation with undiminished severity in one, recurred with increased severity four years later in the other, one of the earliest patients in this series, and was not controlled by what is now regarded as quite inadequate treatment. There are thus 36 patients with rotational vertigo and 38 with positional vertigo. By means of mixed therapy, both niacin and riboflavin being given, rotational vertigo has been relieved in 31 and has been improved and is improving in 5; positional vertigo has been relieved in 23, has been improved and is improving in 14 and was unchanged in 1 (the

second of the 2 patients who had undergone section of the eighth nerve). The duration of these results extends over a period of from six to fifteen months.

In the early days of this investigation treatment was restricted to individual fractions. If the result of the histamine skin test was on the negative side (large negative), the deficiency was assumed to be preponderantly of niacin, and therefore nicotinic acid alone was administered. This served in every case to control the rotational vertigo. Then riboflavin was added (not substituted), and gradually the positional attacks also would be brought under control. Cases 1 and 2 afford examples of this method. On the other hand, when the result of the histamine skin test was on the positive side (small positive), the deficiency was assumed to be preponderantly of riboflavin, and therefore riboflavin alone was administered. By this means positional vertigo could usually be controlled, but rotational vertigo remained untouched and even became more severe. When nicotinic acid was added (not substituted), the rotational vertigo also was brought under control.

Some of the patients in this group had been treated with nicotinic acid before the present investigation started, and their rotational vertigo had been controlled thereby, but they continued to have momentary positional vertigo. Others returned in relapse, but now with attacks of positional type, a change on which they would sometimes comment themselves, saying, "These attacks are different. There is no spin, and I don't vomit any more." These new attacks were always rapidly controlled by the addition of riboflavin.

Thus the results of using individual fractions seriatim in this mixed group have agreed with the results observed in the previous two groups, in which the result of the histamine skin test was either definitely negative or definitely positive, and in each of which one variety of vertigo dominated the clinical picture. Each variety could be abolished by use of the appropriate vitamin fraction. As it became clear that this intermediate group did undoubtedly consist of patients having a more or less evenly distributed mixed deficiency, the members of it began to be treated with both fractions simultaneously, with the result that both types of vertigo could be controlled at the same time. Intravenous injections of a mixture of riboflavin and nicotinic acid or nicotinamide have been given, as already described, supplemented with oral administration, the proportion of each fraction varying according to the clinical picture, the physical signs and the result of the histamine skin test. By this means more rapid and more effective control has been achieved.

Case 9.—The patient, a woman aged 59 years, was first seen in April 1947. From 1934 to 1944 she had experienced at intervals of twelve months or so severe attacks of rotational vertigo with vomiting, both room and bed seeming

to spin to the left or sometimes to roll over backward. In 1944 the character of the attacks changed and they became more frequent. In these later attacks the floor seemed to fall away, and on two occasions she was thrown out of a chair. There was no nausea or vomiting with these attacks. In addition she had frequent flashes of vertigo daily and short-lived waves of unsteadiness. There was considerable loss of hearing in the left ear (35 per cent) with hyperacusis, so that music, voices and loud noises distressed her greatly. In this ear tinnitus had been present off and on for fifteen years but latterly had become constant and was at times so intense that she said it could not have been endured for long at that intensity. In 1940 she had been given histamine intravenously, and thereafter she had an interval of two years free from attacks. In August 1945 she again was given histamine intravenously, and by October her symptoms were bad again. The treatment was repeated, and she was free until February 1947, when a third course of intravenous injections of histamine failed to have any effect. She was then given injections of nicotinic acid, both by vein and by muscle, with considerable flushing reaction and no noticeable improvement. When seen she had been having frequent severe attacks of positional type, in one of which she fell, and she showed signs of severe chronic deficiency of niacin, riboflavin and thiamine. The histamine skin test gave an intermediate response. A course of intravenous injections was started, the doses being gradually increased until by the eighth injection she was receiving: nicotinic acid, 40 mg.; nicotinamide, 400 mg.; riboflavin, 10 mg.; thiamine hydrochloride, 100 mg. This dosage was given daily for ten days and then on alternate days for three months, by which time she had had fifty-three injections in all. In mid-August she reported that she had had two mild "flashes" in July and none since. She had had no tinnitus for four of the previous six weeks, and the hearing of the left ear had returned to normal except for the three highest tones. During all this time she was daily taking supplementary vitamins by mouth, viz.: nicotinamide, 800 mg.; riboflavin, 80 mg.; thiamine hydrochloride, 160 mg. This improvement has continued. On Dec. 1, 1947 she had had no vertigo of any kind since July. She was continuing religiously with her oral therapy and adding an occasional intravenous injection when she felt a little "let down."

THIAMINE DEFICIENCY

In addition to the well recognized symptoms of Ménière's syndrome, symptoms generally accepted as being produced by thiamine deficiency are frequently encountered (tables 1, 2 and 3). Of these the most usual are excessive fatigue, "weak spells" and nervousness. These can be abolished by the exhibition of thiamine (cases 1 and 2). It has therefore become routine to add thiamine to the other fractions in all cases, both by injection and by mouth. Not only does this relieve specific symptoms, it also seems to potentiate the action of the other fractions, as was pointed out by Harris and Moore [28] in 1940 with respect to nicotinic acid as used in the treatment of Ménière's syndrome.

The dosage of thiamine varies according to the individual case but must in general be high as is the case with the other fractions. By injection thiamine hydrochloride has been given in doses of from 25 to 150 mg. daily; orally it has been taken in amounts of from 40 to 400 mg.

daily. In a few exceptional cases even as much as 800 mg., taken daily by mouth, has been necessary for a time to gain control of symptoms.

DOSAGE AND OVERDOSAGE

It will be noticed that the doses used to obtain the results described in this article are considerably larger than those usually advocated even for therapeutic purposes. They have been arrived at as the result of experience. When this investigation started, much smaller doses, in line with those generally accepted, were used, but it was soon found that while a few patients responded to such doses, most did not or did not satisfactorily. Gradually, therefore, the doses administered both parenterally and by mouth came to be increased, and this tendency toward larger doses continues.

A warning is necessary with regard to high dosage—it must be arrived at gradually. If large doses are given immediately or arrived at too rapidly, symptoms may be temporarily increased and attacks provoked. This applies particularly to cases of severe chronic deficiency. The longer the attacks have been in existence and the more severe the signs of chronic deficiency, the more gradual must be the increase, but the higher it may eventually need to be and the longer it will need to be continued if relapses are to be prevented. In any event control is not to be expected immediately, and occasional relapse in the early days of treatment is to be anticipated. Of both these eventualities patients should be warned at the outset. The most that can be offered is a gradual diminution of the frequency and the severity of attacks, and relapse is to be regarded by both patient and physician as an indication for more intensive treatment, not for a throwing up of hands in despair. Treatment of this condition demands patience and persistence on the part of both patient and physician. There are no miraculous cures, and in cases of long standing progress may be discouragingly slow. A patient of 63, with a bald tongue, was under treatment with nicotinic acid and thiamine or five years before her major rotational attacks were brought under complete control, and she has now had none for two years. She continued to have minor positional attacks with periods of headache, distressing tinnitus and general ill health. These have only been reasonably controlled, and still not completely, after two years of added riboflavin and mixed vitamins. In the cases which are not of such long standing in terms of age of patient and duration of symptoms, with more or less acute signs imposed on a mild chronic background, the disease is more rapidly controlled and with smaller doses, but even in these cases no dramatic recovery may be expected, though occasionally one occurs.

The method adopted has been to start with small doses parenterally and orally and to increase the doses slowly until the acute symptoms,

the attacks, are controlled. It has been found inadvisable to begin with a large dose as is usually done in treating the acute stage of a nutritional disorder, owing to the possibility, already mentioned, of increasing symptoms temporarily. This is the more to be avoided because many of these patients display considerable anxiety, particularly those who have been treated by various methods for long periods without success, and to increase symptoms severely in the early stages of treatment is to invite loss of confidence.

Once control has been firmly achieved, parenteral administration can be omitted, but oral dosage must be maintained for months and years if recurrences are ultimately to be prevented. Relapse demands increase of dose and preferably a temporary return to parenteral administration. It must be insisted that there is no specific adequate dose of any or all of the fractions for all patients. The level and the proportions must be tailored to fit the needs of the individual patient, and in the mixed cases a careful balance of all fractions must be kept.

The estimate of adequate dosage must depend on clinical acumen and experience. When the maintenance oral dose appropriate to the individual patient has eventually been arrived at, it must be continued, as cautioned in a previous paragraph, for month after month, year after year. Such long-continued administration is necessary because what has to be done is to repair damaged tissue, and the rebuilding of tissue can obviously come about only slowly. Indeed, some degree of irreversible change may have taken place, and it may well turn out, therefore, that some patients must continue to take small doses of vitamin supplements for the rest of their lives in order to insure the best possible functioning of such tissue as remains.

COMMENT

The Clinical Picture.—That the clinical picture of Ménière's syndrome is more complex than it is generally considered to be was first suggested by the observation of Lermoyez,[5] who as long ago as 1919 described a group of cases characterized by deafness and tinnitus which accompanied vertigo and which were relieved or improved by it—*"le vertigo qui fait entendre"* (the vertigo which restores hearing)—in contrast to the more usual variety in which deafness and tinnitus precede the attacks of vertigo and are made worse by them. The validity of this observation was reenforced by observations that the two groups could be differentiated by their different response to the intradermal injection of histamine, that they presented different clinical pictures and that the Lermoyez picture was comprised in the histamine-

5. Lermoyez, M.: Le vertige qui fait entendre, Presse méd. **27**:1, 1919.

positive group. The present investigation has carried the clinical differentiation of the groups still further.

There are two distinct and characteristic types of disturbance. In one the deafness and tinnitus appear gradually, precede the onset of vertigo even by many years and are increased by the attack, and the vertigo is of rotational type, usually accompanied by vomiting and often by diarrhea. This group gives a small response to an intradermal injection of histamine, has a primary vasoconstrictor mechanism and presents predominantly the signs of niacin deficiency. In the other the deafness and tinnitus appear at the time of the first attack of vertigo, are abolished by the attack in the beginning, though in the course of time there remains a gradually increasing residuum, and the vertigo is of the positional type, accompanied by nausea though seldom by vomiting. This group gives a large response to an intradermal injection of histamine, with pseudopodia, has a primary vasodilator mechanism and presents predominantly the signs of riboflavin deficiency. One common variant of the clinical picture in this group is seen in the patients in whom deafness and tinnitus arise abruptly and severely with the first attack of vertigo and are unrelieved by it—in whom loss of hearing of the affected ear may even be complete and permanent. These are the patients who go to bed hearing, awake in the morning deaf, with roaring tinnitus, get out of bed and fall to the floor. The other variant is seen in the patients in whom vertigo precedes the onset of deafness, sometimes by years, a variant which has been given the unfortunate label of "pseudo-Ménière's disease." The symptoms of these patients correspond closely with those described recently by Kuilman [6] and de Raadt [7] as having occurred among prisoners of war in Batavia and ascribed by them to riboflavin deficiency. Positional vertigo was the characteristic feature; impairment of hearing was unusual. Stannus [8] has described a similar syndrome which he believes to be due to riboflavin deficiency.

Thus it becomes more and more apparent that there are two distinct types of clinical picture comprised by the term Ménière's syndrome. Moreover, evidence begins to accumulate that both are due to vitamin deficiencies—each to a different one. The first is a simple picture due to niacin deficiency; the second, a more complicated one, is due to riboflavin deficiency. What has made for confusion is that both pictures are frequently combined in the same patient, which

6. Kuilman, J.: Camp Vertigo, Nederl. tijdschr. v. geneesk. **90**:983 (Aug. 17) 1946.

7. de Raadt, O. L. E.: Pellagra in the Oto-Neurology and Rhino-Laryngology, Leiden, Universitaire Pers Leiden, 1947.

8. Stannus, H. S.: Disorders of the Nervous System Due to Malnutrition, Brit. M. J. **1**:342 (Feb. 21) 1948.

is entirely understandable if the theory of Ménière's syndrome as a deficiency disease is correct, since vitamin deficiencies seldom, if ever, appear singly. These findings explain why no one therapy is effective in every case and why one form of treatment is seldom completely effective in any.

Nicotinic Acid vs. Nicotinamide.—Nicotinic acid has proved successful in the treatment of the vasoconstrictor group. It was originally selected because of its vasodilator effect, but the possibility of its exerting a vitamin action has always been present. This possibility, however, seemed to be ruled out because of the apparent ineffectiveness of nicotinamide in controlling attacks in this group. The ineffectiveness is quite comprehensible if the disturbance is a purely vascular one, incomprehensible if it is due purely to a deficiency, since the amide should then be as efficacious as the acid. The explanation of the apparent anomaly is that both conditions obtain. The mechanism of the attack in this group is a vasospasm, but the cause of the vasospasm is a vitamin deficiency. Thus nicotinic acid produces more satisfactory results in the vasoconstrictor group than the amide because it combines both functions, that of vasodilator and that of vitamin.

Actually it is possible sometimes to control attacks with nicotinamide. Four patients belonging to the vasoconstrictor group have been treated successfully with nicotinamide alone, but they required more prolonged and more intensive treatment and much larger doses to obtain control than have ever been found necessary with the acid. Failure to appreciate this has been the reason for failure to obtain control with the amide in the past.

For instance, a woman aged 52 years, who complained of moderately severe attacks, occurring once every two or three weeks for a year only, was treated throughout with the amide. She required forty-seven intravenous injections of 400 to 600 mg. each of the amide over a period of four months, given daily at first, then three a week and finally two a week, before satisfactory control was established. At the same time she took 800 to 1,600 mg. by mouth daily. Under these dosages she had two mild attacks only in the final two months of the injection treatment and has been kept free of attacks with oral medication but requires 1,600 mg. of nicotinamide daily to prevent relapse. This is a large dose. Experience with nicotinic acid has been that the requirement of an intravenous dose of as much as 100 mg. is rare, the average control dose ranging around 75 mg. intravenously and 400 mg. orally.

Thus, while it is possible to establish control with the amide, the process is much slower than with the acid, requires a considerably higher level of doses and is much more expensive. Some patients who have previously received the acid and subsequently been given the amide

ask to return to the acid because they feel much better with it. One such patient who had been treated previously and successfully with 50 mg. of acid intravenously and 200 mg. orally daily had failed to keep up his oral maintenance dose and returned in relapse. He was given the amide instead of the acid, but after four daily doses of 200 mg. intravenously and 800 mg. orally he asked for his "old" medicine again, as he had felt so much better with it than with the amide.

Once control has been established with the acid it can frequently be maintained with the amide if desired. Even so, the vasodilator effect of the acid is preferable until all vasoconstrictor disturbance has been overcome. This change can be determined by the histamine response, which changes from the small to the intermediate appearance.

What has been said in the preceding paragraphs applies to the vasoconstrictor or histamine-insensitive group. In the vasodilator or histamine-sensitive group, however, and in those members of the intermediate group which veer toward the histamine-sensitive end of the scale, the amide is mandatory in the treatment of the niacin deficiency. It has been a repeated experience, and one should emphasize it again, that if members of the vasodilator group are given nicotinic acid they are made worse as regards both frequency and severity of attacks. To give one dramatic example out of many less dramatic, a member of this group was on one occasion given by mistake nicotinic acid intravenously instead of histamine subcutaneously. Fifteen minutes later she had an attack which threw her violently to the ground in the worst attack she had ever experienced, luckily without serious injury. It cannot be too strongly insisted that nicotinic acid with its vasodilator action is poison to the vasodilator group. For the treatment of niacin deficiency in this group the amide, and the amide only, must be used because it has no vasodilator effect.

Riboflavin and Histamine.—What is the connection between riboflavin and histamine? That there must be some connection seems certain from the consistency with which riboflavin deficiency is found in association with a positive response to injected histamine and from the fact that either substance will relieve symptoms in this group. What the connection may be is at present not known. Zeller and his co-workers [9] have reported that histaminase is a flavoprotein, though other workers [10] have contradicted this. If it were so, it might account for the beneficial action of histamine in riboflavin-deficient subjects by encouraging the production of histaminase. Against this possibility is

9. Zeller, E. A.; Stern, R., and Wenk, M.: Ueber die Diamin-Diamin-Oxydase Reaktion: VI. Mitteilung über den enzymatischen Abbau von Poly-aminem, Helv. chem. acta **23**:3, 1940.

10. LeLoir, L. F., and Green, D. E.: Histamine Oxidase, Federation Proc. **5**:144 (Feb.) 1946.

the fact that commercially available histaminase has proved completely ineffective in treatment.

Whatever the explanation, the clinical fact remains that the histamine-sensitive patients show signs of riboflavin deficiency and can be relieved temporarily by the administration of either histamine or riboflavin. It is hoped that the prolonged use of riboflavin will ultimately give permanent relief in this group, in the same degree that nicotinic acid does in the vasoconstrictor group, but the patients have as yet not been observed long enough to establish this. What has been established is that attacks can be controlled with riboflavin if this is administered in suitable dosage. Two patients not included in this series, one a physician, have on several occasions staved off what they knew would have been severe attacks with a large dose of riboflavin.

Dietary vs. Nutritional Deficiency.—This concept, that Ménière's syndrome is a nutritional disturbance, may be objected to on the grounds that the subjects of Ménière's syndrome as seen in everyday practice do not, in most cases, subsist on a frankly inadequate diet. However, if a careful dietary history is taken, it will usually be found that their dietary habits are bad and that their present diet is unbalanced, lacking in important foods, such as milk, and all too frequently improperly prepared. This has been pointed out by Selfridge.[11] Moreover, not infrequently their childhood feeding has been inadequate as the result of economic or cultural factors. Because of this and of other later incidents which have interfered with nutrition, such as a severe illness, alcoholism or gastrointestinal disurbances interfering with adequate absorption, there has been built up over the years a condition of chronic nutritional deficiency which is too severe to be corrected by diet alone no matter how adequate that diet may be. It is this chronic background which is exhibited by the patients with Ménière's syndrome.

The story, then, is this: For one reason or another a state of chronic nutritional deficiency arises which is continuously increased by the stresses and strains of living. Eventually comes a time when the subject is teetering on the edge of a nutritional precipice, conscious perhaps of vague ill health, though without the symptoms of a defined disease, until one day some perhaps trivial incident, a cold, a tooth extraction, a minor accident, topples him over into the abyss of an acute deficiency with full-blown overt, even catastrophic, symptoms. He now has an acute deficiency imposed on his background of chronic deficiency. If the picture is one which is recognized as nutritional in origin, such as pellagra, he will be treated accordingly and his acute symptoms will disappear. But unless the chronic background is also treated over a long period, his acute symptoms will recur as a result of smaller and smaller insults and,

11. Selfridge, G.: Ménière's Symptom Complex: Its Relation to Chemistry, Arch. Otolaryng. **49**:1 (Jan.) 1949.

therefore, at shorter and shorter intervals, in fact at any time when he is subjected to undue stress. It is the failure to treat adequately, or at all, the chronic background which accounts for the notorious tendency of nutritional diseases to relapse.

If the classic picture of Ménière's syndrome is considered in this light, it will be seen that it conforms with the picture of a nutritional disorder. Usually the first evidence of trouble observed by the patient is the gradual onset of deafness with tinnitus, though, if he be closely questioned, he will often admit that he was conscious at this time, or even before he was aware of any cochlear disturbance, of a state of health below normal—he tired easily, he was lacking in ambition, he was nervous and irritable, his digestion was not as good as it should have been. Then one day he "caught a cold" or had an attack of "grip" and immediately thereafter he had his first attack of vertigo. How often that or some similar story is told and how often it is disregarded! The attack of vertigo puts him to bed, forces him to give up work for a time or to take life more easily and he builds up a small reserve again. When that reserve is exhausted—how soon depends on the degree of his deficiency, on the degree of impairment of function of his tissues—comes another attack, another acute breakdown. So the disease goes on in the familiar cycle of remission and relapse, with increasing frequency of attacks, increasing evidence of impairment of general and cochlear health, with increasingly severe chronic deficiency. When on top of this are imposed well intentioned but ill advised dietary restrictions the result may be disastrous. Such a case has been investigated recently. A man in whom attacks had increased up to two or three a week was admitted to a hospital where he was fed a severely restricted diet, with the result that he lost 30 pounds (14 Kg.) in six weeks. Attacks gradually increased during this time until he was having three or four a day, was constantly unsteady and was alarmingly ill. Intensive vitamin therapy controlled the attacks in a week, and at the time of writing he has been without further attacks for five weeks, has regained his sense of balance, is putting on weight and looks and feels like a different person. Such improvement in general health is a fairly constant happening in cases in which the patient is under adequate nutritional therapy and one on which the patient is often the first to comment.

SUMMARY

The present investigation has brought out the following points:

1. Subjects of Ménière's syndrome experience two distinct types of vertigo, rotational and positional, which have been found to be associated with different and specific vitamin deficiencies.

2. Those patients who experience rotational vertigo alone, show signs of nicotinic acid deficiency in the tongue, give a small response to histamine injected intradermally and can be relieved of their attacks with nicotinic acid.

3. Those patients who experience positional vertigo alone, show signs of riboflavin deficiency in the tongue, the eyes and the skin, give a large response to histamine injected intradermally and can be relieved of their attacks with riboflavin.

4. Those patients, the majority, who experience both kinds of vertigo, show signs of a deficiency of both fractions, give an intermediate response to histamine injected intradermally and can be relieved of their attacks only if both fractions are exhibited.

5. Other symptoms common in patients with Ménière's syndrome can be related to other deficiencies, such as the fatigue, irritability and palpitations associated with thiamine deficiency, and can be relieved by administration of the appropriate vitamin.

6. Successful therapeutic results depend on a method of treatment and a level of dosage such as have been described.

310 Lexington Avenue.

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ESOPHAGEAL VARICES IN BANTI'S DISEASE

Report of Two Cases in Which the Condition Was Improved with Injection of Sclerosing Solutions

ROBERT B. LEWY, M.D.
CHICAGO

ANY UNDERSTANDING of the problem of esophageal varices requires some knowledge of the anatomy of the portal vein and its collateral veins. It is because of the gradual occlusion of this vein and the forced development of the collateral system that these varices occur.

The portal vein which courses through the liver represents a confluence of the superior mesenteric vein, the inferior mesenteric vein, the splenic vein and the coronary, cystic and parumbilical veins and their tributaries. The portal system may be defined to include all the veins that drain the abdominal portion of the digestive tube.

The cause of the esophageal varices is the same as that of some varices elsewhere in the body, e. g., hemorrhoids, rests in the mechanical obstruction of the portal vein. Any of a series of possible causes which would effect a gradual closure of this venous system and which permitted time for the development of the collateral system might thus be the responsible factor.

The collateral circulation that develops could not do so but for preexisting communications. These are between the gastric and the esophageal veins, the veins of the colon and the duodenum and the left renal vein (the so-called accessory portal system of Sappey—in which branches pass around the falciform ligament to unite with epigastric and internal mammary veins). There is also a large single vein, the parumbilical, which may pass from the hilus of the liver to the umbilicus. (When this functions it produces the well known caput medusae.) The third is that of veins of Retzius, which may connect the intestinal

From the Department of Otolaryngology, University of Illinois College of Medicine and Veterans Administration Hospital, Hines, Ill.

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veins with the inferior vena cava and its retroperitoneal branches; the fourth and last channel of communication is between the inferior mesenteric vein and the hemorrhoidal veins. These open into the hypogastric vein.

As indicated by the expression "gradual occlusion of the portal system," the chief causes of chronic and long term portal obstruction rest within the group of pathologic conditions known as cirrhosis of the liver. All types of cirrhosis of the liver have one feature in common, an increase in the connective tissue of that organ. When and if this connective tissue is in a position to restrict the return flow of blood of the portal system, the opportunity for the setting up of the collateral venous drainage is created.

In the cases to be reported one is especially concerned with Banti's disease, known also as splenic anemia. This is a chronic disease of unknown origin and cause. The origin has been described by some vaguely as toxic and primary in the spleen and by more modern authors as primary in the liver. It is characterized by splenomegaly, anemia, leukopenia and a "tendency to gastric hemorrhages." The later stages of the disease are characterized by cirrhosis of the liver, jaundice and ascites. The term "Banti's disease" is usually reserved for the late stage when the cirrhotic elements have supervened. This condition is usually considered a disease entity. By some it is considered a syndrome.

Banti divided the disease into three period phases. The first phase is described as the anemic phase, the second as the intermediary and the third as the cirrhotic. The first stage usually lasts for two or more years. The second stage lasts for a few months and the third to the end, usually to terminate with an intercurrent infection or a hemorrhage.

Primary treatment is exclusively surgical. It is splenectomy. This results on occasion in complete relief; in some cases, in marked relief. Recently, anastomosis of the splenic and the left renal vein, combined with nephrectomy, has been performed to create a bypass. Secondary treatment is palliative and supportive. Transfusion and abdominal paracentesis may be required in the cirrhotic stage.

Other cirrhoses should be mentioned. These may be classified into three general groups: (1) toxic cirrhoses (the most important of which is alcoholic); (2) cirrhoses from chronic congestion of the hepatic blood vessels, as in chronic failing or noncompetent right side of the heart; (3) cirrhosis from chronic obstruction of the bile ducts (this group would include, in addition to Banti's disease, syphilitic cirrhosis and the hypertrophic cirrhosis of Hanot). In rare instances cirrhosis due to malaria has been reported.

The reports of Kirklin and Moersch,[1] Moersch [2] and Wolf [3] represent the most extensive pioneering work that has been done on the subject of the sclerosing treatment of esophageal varices. The first report in the Moersch series and the first case reported in this country presents a patient with a past history of hematemesis and melena of several years' duration. Examination of the patient revealed an enlarged

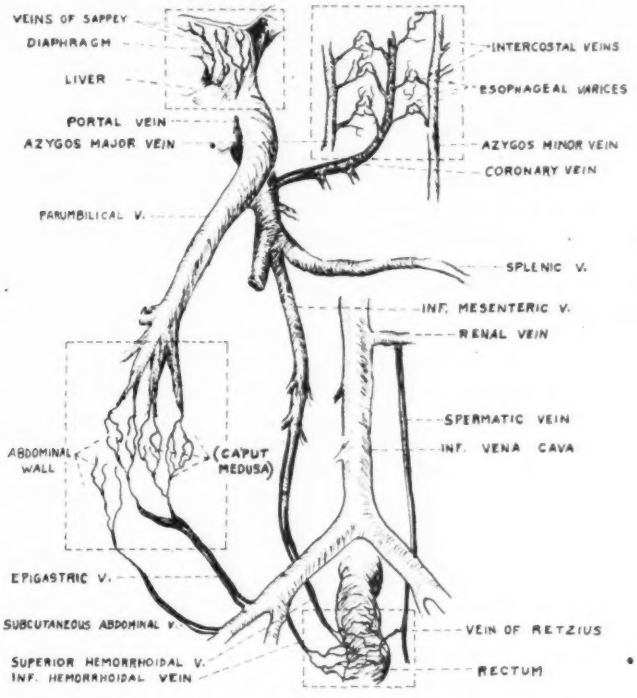

Fig. 1.—The portal circulation with collateral shunts (after Moersch).[2]

spleen and secondary anemia. On roentgenologic study of the esophagus it was noted that the lumen appeared, at a point just below the

1. Kirklin, B. R., and Moersch, H. J.: Report of a Case of Roentgenologically Demonstrable Esophageal Varices Complicating Splenomegaly, Radiology **17**: 573 (Sept.) 1931.

2. Moersch, H. J.: Treatment of Esophageal Varices by Injection of Sclerosing Solution, J. Thoracic Surg. **10**:300 (Feb.) 1941.

3. Wolf, G.: Die Erkennung von Oesophagus-Varizen im Röntgenbilde, Fortschr. a. d. Geb. d. Röntgenstrahlen **37**:890 (June) 1928.

aortic arch, to be a narrow and tortuous canal with marginal defections. These defections were a group of multiple intrusions, and when barium sulfate mixtures infiltrated between them they depicted a lumen of normal width. The diagnosis was thus accurately established with this and endoscopic observation.

In his second report Moersch [2] stated that he had treated esophageal varices by injecting a sclerosing solution. In this report is the first record of such work in this country. Also included is a reference to the earliest known diagnosis of esophageal varix, the report of Power in 1839. Moersch stated that these varices are not too uncommon. They occurred in 5 per cent of 688 patients who came to the Mayo Clinic complaining of hematemesis. In this group of cases of varicose veins Banti's disease was the most frequent cause.

In a later report Moersch [4] recorded the treatment of 10 additional patients. These were usually treated with the sclerosing solution after splenectomy, as splenectomy was considered essential to any prospect of cure of the disease; this because, among other results, the removal of the spleen, if effective, reduces by 20 per cent the amount of blood that passes through the portal system. Moersch has injected as much as 9 cc. into patients without ill effect. He stated that the injection may be accompanied by moderate to severe pain. The whole group of patients, on later canvassing, reported that their condition, was much improved and that they had no more bleeding. The Moersch technic was to inject 0.5 cc. of a 2.5 per cent sodium morrhuate solution by way of an esophagoscope and long 25 caliber needle. The latter necessarily has a length greater than the scope. Injections were usually done partially and at intervals of three to four days.

Welt and Blatteis [5] reported a case in which they used a sclerosing solution. Their patient had been operated on previously for appendicitis and had had a stormy postoperative course. As a result (perhaps of adhesions), the patient had six subsequent operations. After hematemesis and tarry stools had occurred, the presence of esophageal varices was suspected. This was verified with roentgenologic studies. Bleeding was so severe that the patient had required seven transfusions. At esophagoscopic examination the lumen was seen to be two thirds obstructed near the base by enlarged veins. The largest of these were at 12 and 6 o'clock. Two cubic centimeters of a solution of sodium morrhuate was injected. Further observation revealed great shrinkage of the varices and no further bleeding.

4. Moersch, H. J.: Further Studies on the Treatment of Esophageal Varices by the Injection of Sclerosing Solution, Ann. Otol., Rhin. & Laryng. **50**:1233 (Dec.) 1941.

5. Welt, B., and Blatteis, S. R.: Esophageal Varices: Case Report, Am. J. Surg. **63**:415 (March) 1944.

Proper control of esophageal varices is absolutely essential. Fatal bleeding can and does occur. One such case was reported in the *New England Medical Journal*[6] of July 1945; another was reported from Australia by Duffy and Fraser[7] in March 1944.

The only other past attempts at therapeutic control of this problem that had any success were of a more difficult and arduous type. One case was reported in which the coronary vein was ligated and retrograde injection of a sclerosing solution was done by a transabdominal approach. The retrograde flow of the solution occluded the esophageal veins.

The most recent report on this subject in the literature is that of Patterson and Rouse.[8] They included in their report the histories of 8 patients of a total of 12 whom they had treated for this condition. The youngest was aged 3 and the oldest 66. A total of three to four injections was usually employed, and the intervals between "sittings" varied from a week to a year. One patient received injection before and after splenectomy; 4, after splenectomy; 7, before splenectomy. There were two fatalities in their series; both were from hemorrhage, and one of these occurred some time after the procedure.

Herein is reported for the record the histories of 2 patients treated by the method of Moersch, with recovery.

REPORT OF CASES

CASE 1.—R. G., a white man aged 23, was inducted into the military service and was shipped to the Southwest Pacific theater. He remained there and in good health until April 1944, when he had chills and fever. A series of smears of the blood were studied for malaria but were all negative. During the period of hospitalization he had two episodes of vomiting. Each time he lost about a half cup of dark blood. Shortly after hospitalization an enlarged spleen was noted. He remained hospitalized and was sent back to the United States, where he was admitted to Vaughan General Hospital for definitive treatment. A diagnosis of Banti's disease was made. On Dec. 5, 1944 splenectomy was performed. After surgical recovery he was discharged from the hospital. He remained in fair health until May 15, 1946. At this time he became nauseated and vomited a pint of blood. That night he fainted. As a result of this acute episode he was admitted to the Veterans Administration Hospital at Hines, Ill.

At admission the physical examination revealed a well developed and well nourished white man moderately ill. The positive physical findings otherwise were grade II systolic murmur over the precordium and a scar over the left upper quadrant of the abdomen. The red blood cell count was 2,350,000 and the white

6. Portal Cirrhosis and Rupture of Esophageal and Gastric Veins, Cabot Case 3128, New England J. Med. **233**:48 (July 12) 1945.

7. Duffy, D. G., and Fraser, A. N.: Fatal Bleeding from Varicosities, M. J. Australia **1**:202 (March 4) 1944.

8. Patterson, C. O., and Rouse, M. O.: The Injection Treatment of Esophageal Varices, J. A. M. A. **130**:384 (Feb. 16) 1946.

cell count 14,800; the hemoglobin content was 51 per cent. After a short period of hospitalization the red blood cell count rose somewhat. It was suggested that he was bleeding from esophageal varices, and it was decided to perform an esophagoscopic examination. This was done on June 29. Varicose veins were visualized, most marked at the cardial end of the esophagus. Esophagoscopic examination was again performed, October 8, and a 2.5 per cent sodium morrhuate solution was injected into the varices. Esophagoscopic examination was repeated on December 6, and the previously treated varices were found to be flattened. On December 7 endoscopic examination was again performed and the sclerosing solution injected into varices in the anterior wall. The varices seemed to have less volume than

Fig. 2.—Oblique roentgenographic view of the chest demonstrating a defect of the esophagus caused by the intrusion of a varix (indicated by an arrow).

before. After a period of no hemorrhage the patient was discharged to his home. A follow-up letter brought the reply that he has had no symptoms of the varices in the three months since his discharge. He has had no tarry stools, no vomiting of blood, no red blood in the stools, no difficulty in swallowing. He has been working part time.

Case 2.—R. Mc., a white man aged 38, was admitted to the Veterans Administration Hospital at Hines, Ill., in 1941, with a condition diagnosed as Banti's disease. His occupation was that of truck driver. His habits included consumption of

moderate amounts of alcoholic beverages and tobacco. There was a previous history of syphilis and gonorrhea. He had been given a similar diagnosis in the past and had undergone splenectomy. He was discharged after a short period of hospitalization and readmitted in June 1946 because of bleeding believed to be from esophageal varices.

The positive physical findings at the time of admission were a soft systolic murmur over the apex and a midline scar. There was rather marked pallor. Repeated counts of the red blood cells revealed usually 3,000,000. Whenever the level reached 3,300,000 he bled.

The interim history revealed that he had had his first bleeding in the fall of 1937 and that he had been hospitalized more than 10 times. Roentgenographic studies revealed a slight irregularity of the lumen of the esophagus at its lower end. The roentgenologic diagnosis was esophageal varix; it was confirmed by endoscopic examination.

The diagnosis was confused at first by not finding any varices on endoscopic examination, Oct. 25, 1946. However, this procedure was repeated, and on October 22 two varices were found and a sclerosing solution was injected. One of these was half way down the esophageal wall at 12 o'clock; the other near the cardia. The procedure was repeated on November 5 and a sclerosing solution injected into a varix just above the cardiac rosettte at 5 o'clock. From the second injection severe pain resulted, which required morphine for several days. After this second injection he had no more episodes of bleeding. Some months later a follow-up letter was sent, and the following reply was noted: He had vomited no blood since his discharge from the hospital. However, he had black tarry stools on two occasions in 1946 and on one in January 1947. He also had hemorrhoids and attributed some red blood in the stools to this source. He felt fairly well. The red blood cell count was near 3,500,000. He was able to walk and drive a car. He was admitted to another hospital, where two transfusions were given. He had at this time considerable fluid in the abdominal cavity, which was drained off. He stated that he felt that the procedure did not get all the leaky veins and that more work might have to be done eventually.

COMMENT

The results recorded in the medical literature and the 2 cases herein reported lead to one conclusion, that the injection procedure is one of relative safety and represents a more reasonable course of action than doing nothing or making a much more radical approach to this type of varix by a transabdominal route. The principle that the simplest and safest procedure that is effective should be the one of choice seems in this condition most demanding. The transabdominal surgical alternatives are the ligation of the coronary vein or the ligation of this vein combined with a "retrograde" injection of the collateral vessels. In this way the sclerosing is accomplished from below rather than from above.

In all previous cases reported in this country as well as in those herein reported the procedure has proved to be innocuous. The worst immediate complication has been pain. There have been late failures but no late complications. The "failures" may have been from missing small

varices at the time of the original injections. They represent a small percentage of the total number of patients treated. I have found no comparable record of effectiveness and safety with the other surgical solutions.

Possible complications include hemorrhage at the time of injection, necrosis and perforation of the esophagus, ulceration of the esophageal wall and hemorrhage from misplaced sclerosing solution. None of these are recorded as the experience in those cases previously reported or in these 2.

The chief indication for the procedure becomes clear from the substance of this and previous reports. It is the presence of esophageal varices with or without hemorrhage, obstruction or dysphagia. Contraindications depend on judgment, balancing the threat of the varices and the condition of the patient. As nonabsolutes, advanced cardiac disease, advanced age, terminal state and marked hypertension should be considered. Also to be listed are anatomic deformities of the spinal column, the mouth or the jaw that would render endoscopic examination impossible. The technic is that of cautious endoscopic examination. It is better to accomplish the result in several sittings, if the introduction of the scope presents no difficulties, than to try to accomplish all at once. No stronger than 2.5 per cent solution of sodium morrhuate should be used, and every patient should have a prior skin test for sensitivity to this drug. Adequate preoperative sedation is mandatory. The base of the varix should be engaged in the lip of the scope to fix it. Pressure should be continued to localize the area of reaction. The patient should be in an exaggerated Trendelenburg position. This should tend to cause the sclerosing material to flow toward the head and thus act on a larger segment of the vein.

The unfinished nature exhibited in 1 of my patients and the two late hemorrhages that followed in 2 of the patients of Rouse and Patterson emphasize the necessity for careful, long term follow-up. This should be done for six to twelve months at least. There remains the possibility of recanalizations because of the increased pressure distal to the thrombus in the portal system.

Further studies and observation are required by the internist and the endoscopist in the analysis of this problem. They would like to know what happens to the collateral veins when the esophageal veins are occluded. What should venography show? What happens to the pressure in the portal system, and what effect does this procedure have on hepatic function? Presumably the blocking off of a working shunt around the liver bed should raise pressure and increase or precipitate ascites. Is this presumption correct?

SUMMARY AND CONCLUSIONS

Two cases in which esophageal varices were associated with Banti's disease are reported. In one case an apparent excellent and permanent result was demonstrated following injection of a sclerosing solution. In the second case improvement was shown, but the course indicated that examination and treatment may need to be repeated for a period of at least a year.

The procedure of injection is not difficult for a trained endoscopist. It is simpler and safer than the nonendoscopic methods used heretofore.

A small amount of bleeding should be no deterrent to continuing the procedure. Bleeding is not usually started by this procedure.

25 East Washington Street.

OTOSCLEROSIS ASSOCIATED WITH OSTEOPOROSIS AND LABYRINTHITIS CHRONICA OSSIFICANS

HANS BRUNNER, M.D.
NEWARK, N. J.

THE MICROSCOPIC changes in otosclerosis are well known, although several questions, particularly those concerning the onset of the pathologic changes, remain unanswered. The etiology of otosclerosis, however, is a problem; neither examinations of human temporal bones nor experimental work has succeeded in solving it. For this reason the classification of otosclerosis as a pathologic entity is likewise problematic, and investigations which may eventually shed light on the etiology and the pathologic type of otosclerosis are of interest. An investigation of this sort is based on the question of whether systemic disorders, metabolic or endocrine in origin, or pathologic changes in the entire capsule of the labyrinth exert an influence on the progress of otosclerosis. Some work on the subject is available. For example, Mayer [1] examined a temporal bone presenting an association of otosclerosis and osteitis deformans. I examined a temporal bone in a case of otosclerosis associated with ochronosis.[2] From these and other reports, the conclusion can be drawn that no systemic disorders or diseases of the entire capsule of the labyrinth have been discovered which modify the pathologic changes in otosclerosis.

The following case of otosclerosis and osteoporosis of the capsule of the labyrinth is an additional contribution to the literature. The labyrinthitis chronica ossificans in the inner ear increases the importance of the case.

REPORT OF A CASE

The patient was a 41 year old white man with dementia precox. He was deaf, but he understood lip reading. He died of empyema of the chest. Autopsy revealed empyema of the right pleural cavity, collapse induration of the middle lobe of the right lung and the lower lobe of both lungs, coronary sclerosis, myocardial fibrosis, edema of the left lung and splenitis.

Macroscopic Examination of the Temporal Bones.—The mastoid process on both sides and the apex of the left petrous portion were diploic. The carotid and sigmoid

From the Department of Otology, Rhinology and Laryngology, University of Illinois College of Medicine.

1. Mayer, O.: Untersuchungen über die Otosklerose, Vienna, A. Hölder, 1917.

2. Brunner, H.: Ueber die Veränderungen des Schläfenbeines bei der Ochronose, Monatschr. f. Ohrenh. **63**:997, 1929.

sinuses were normal. At the posterior surface of each temporal bone there were two hyperostoses: One encircled the opening of the internal auditory meatus; the other was located close to the superior angle of the petrous bone and was approximately 1 cm. in length.

Microscopic Examination of Temporal Bones.—The observations were essentially the same on the two sides.

Eustachian Tube, Middle Ear and Mastoid Antrum: The mucous membrane of the tube was slightly thickened, owing to an increase of connective tissue, and was covered with a cubical epithelium. The tensor tympani muscle and the drum membrane were normal. The tympanic cavity was large; it did not contain fluid.

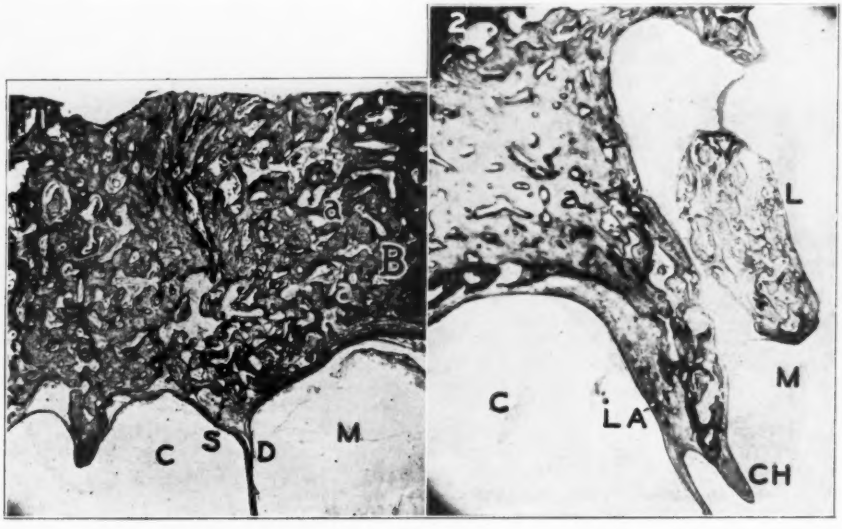

Fig. 1.—*1*, section through the inferior insertion of the drum (*D*), the external auditory canal (*C*), the tympanic cavity (*M*); (*S*) spongy bone (osteoporosis); (*B*) normal bone of the inferior wall of the tympanic cavity; (*a*) boundary between osteoporotic and normal bone. *2*, section through the lateral wall of the attic (*LA*), the incus (*L*), the tympanic cavity (*M*), the chorda tympani (*CH*) and the external canal (*C*); *a-a* boundary between osteoporotic and normal bone.

The mucous membrane of the tympanic cavity was slightly thickened, and the epithelium was flat or cubical. The epitympanum (attic) was large and empty; the tegmen tympani was diploic. The annulus tympanicus, particularly in the area of the lateral wall of the attic, consisted of spongy bone (fig. 1), the trabeculae of which were formed of weblike bone. The bone had a large number of small cavities which did not contain osteocytes, indicating atrophy of the bone trabeculae. Between the trabeculae were numerous marrow spaces filled with connective tissue and capillaries, but there was neither osteogenesis nor osteoclasis. The drum made its insertion at the cancellous atrophic bone (fig. 1).

The malleus and incus presented advanced osteoporosis (fig. 1). There were large marrow spaces, filled with loose connective tissue and hyperemic capillaries. The bone trabeculae were definitely atrophic and frequently presented Howship's lacunas, but there were no osteoclasts. Within the marrow spaces an inconspicuous metaplastic ossification of the connective tissue had taken place. The superior and posterior walls of the external canal and the short process of the incus were of different structure. There was cancellous bone, with small marrow spaces between the bone trabeculae. The latter consisted of weblike bone, rich in osteocytes. The marrow spaces were filled with connective tissue and capillaries. The boundaries of the marrow spaces were covered with a line of blue cement and were aplastic. The stapes was normal. The mastoid antrum was large and did not contain fluid or connective tissue.

Membranous Internal Ear: Although the internal ear was badly damaged in preparation for the examination, the following information was obtained: There was an accumulation of connective tissue in both scalae. Within the connective tissue, occasional small cysts and frequently a network of spicules of calcium or newly formed bone were observed (fig. 2 *1*). On the right side there were adhesions in the scala tympani of the upper portion of the basilar coil and in the lower portion of the middle coil of the cochlea. The adhesions, which included pigment and calcium, pulled the lamina spiralis ossea, as well as the basilar membrane of the cochlea, toward the floor of the scala tympani, causing obliteration of the scala tympani and a compensating dilatation of the scala vestibuli. On the right side there was a septum vestibulocochleare,[2a] which had grown together with Reissner's membrane of the vestibular coil of the cochlea. The endochondral layer of the capsule of the labyrinth, which was in contact with the mass of connective tissue within the cochlea, had a smooth surface; that is, it did not present signs of bone absorption or bone deposition (fig. 2 *1*). At several places, Reissner's membrane had grown together with the connective tissue within the scala vestibuli. This caused a pronounced dilatation of the cochlear canal, except in the lower part of the basilar coil, where the cochlear canal had a normal configuration. The cochlear canal did not contain pathologic fluid or connective tissue. In the larger part of the cochlea, the crista spiralis was greatly lowered; the spiral nerve, the spiral ganglion, the organ of Corti and the stria vascularis were absent, and the ligamentum spirale was much diminished in size. In the basilar coil on the left side, the stria vascularis was present but strongly atrophic.

The modiolus consisted of a small amount of bone trabeculae (fig. 2 *1*). The channels of the modiolus were covered with a thick layer of calcium and contained much loose connective tissue. The number of ganglion cells and nerves was greatly diminished. The blood vessels had very thin walls; they were either obliterated or empty. The amount of pigment was normal on the left side but apparently slightly increased on the right side. The nerves of the internal meatus, except the facial nerve, contained a great amount of degenerated myelin sheaths and connective tissue. The vestibular ganglion appeared normal. The sacculus on the right side was dilated; the walls had grown together with the inner surface of the foot plate of the stapes, as well as with Reissner's membrane of the vestibular coil of the cochlea. The macula sacculi was atrophic on both sides. The cochlear aqueduct on the right side was obliterated by connective tissue. There was definite atrophy of the macula utriculi on the right side. The cristae were too much damaged to permit conclusions regarding them; however, the pigment of the cristae was appar-

2a. Alexander, G.: Labyrinthitis chronica ossificans: Ein Beitrag zur Anatomie der Taubstummheit, Monatschr. f. Ohrenh. **40**:489, 1906.

ently increased. On the left side there was a subepithelial area of hyalin at the slope of the crista of the external ampulla. Within that area there was an accumulation of pigment, which produced slight bulging of the wall of the ampulla toward the lumen. The ampullosaccular channel contained only a small amount of nerve fibers.

Bony Capsule of Internal Ear: In the area of the cochlea the periosteal layer was very well developed. It contained large marrow spaces, which were filled with fat or with lymphatic marrow. The boundaries of the marrow spaces were aplastic, and the bone trabeculae consisted largely of lamellar bone. The marrow spaces extended deep into the endochondral layer and may occasionally have reached the endosteum (fig. 2 *1*). The marrow spaces within the endochondral layer contained fat or connective tissue exclusively; there was no lymphatic marrow. The

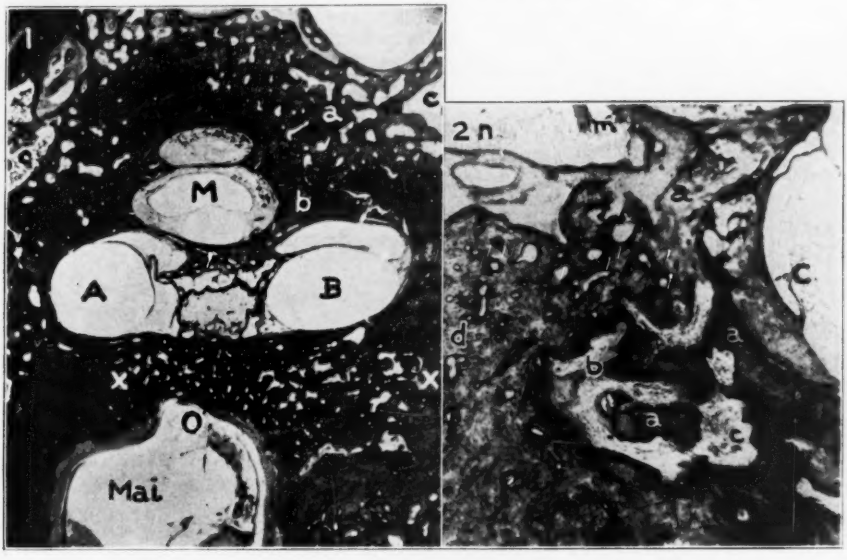

Fig. 2.—*1*, section through the internal auditory meatus (*Mai*) and the cochlea on the left side; (*x-x*) boundary between otosclerotic bone (*O*) and normal bone; (*A*) upper basilar coil of cochlea, damaged by preparation, (*B*) basilar coil, showing absence of organ of Corti, (*a*) osteoporosis, (*b*) remnants of endochondral and endosteal bone, (*c*) large marrow spaces in the periosteal layer. In the tangential section through the middle coil (*M*) and the tip of the cochlea there are newly formed connective tissue containing trabeculae of newly formed bone and narrowing of the lumen of the cochlea, particularly at the tip of the cochlea. Note the smooth walls of the osseous cochlea, the absence of the spiral ganglion and the blood vessels of the modiolus, surrounded by calcium and newly formed bone. *2*, an otosclerotic area at the round window (*m*) and the inferior ampulla (*C*) on the right side; (*a*) remnants of the endochrondal bone of the capsule of the labyrinth, (*b*) osteoid substance, (*c*) connective tissue, (*d*) weblike bone, (*n*) recess of round window.

bone trabeculae in this area consisted of weblike bone, rich in osteocytes. The marrow spaces within the endochondral layer were more pronounced on the left side than on the right.

In the fundus of the internal auditory meatus there was a large area of otosclerosis (fig. 2 *1*). This area consisted either of cancellous bone or of solid, weblike bone, which contained several obliterated blood vessel channels and which showed advanced basophilia. In the latter there were numerous small marrow spaces, which contained connective tissue and capillaries. Within the otosclerotic area there were remnants of the original capsule of the labyrinth, consisting of lamellar bone. These remnants showed many Howship lacunas and were surrounded either by connective tissue of a marrow space or by the weblike bone of the otosclerotic area. In the latter case there was a line of cement between the two types of bone. Where the otosclerotic area was adjacent to the solid bone of the endochondral layer, its boundaries were sharp; where the area was adjacent to the osteoporotic bone of the periosteal layer of the capsule of the labyrinth, its boundaries were less distinct.

In the areas of the saccule and the vestibular portion of the cochlea, the periosteal layer, adjacent to the posterior cranial fossa, had a thickness of 3 to 4 mm.; where it was adjacent to the middle fossa, it was somewhat thinner. It had the same structure as in the area of the cochlea, except that there was connective tissue in several marrow spaces close to the middle fossa. The endochondral layer was normal and contained only a small number of interglobular spaces. In front of the oval window there was an otosclerotic focus, consisting of trabeculae of weblike bone and of small marrow spaces, filled with connective tissue and hyperemic capillaries and having aplastic boundaries. The otosclerotic focus had sharp boundaries toward the endochrondal bone, while the boundaries toward the periosteal bone were less pronounced.

Toward the utricle and the semicircular canals the otosclerotic area increased rapidly in size, particularly on the right side (fig. 3). Ultimately, the otosclerotic area had encircled the entire utricle and the vestibular portion of the cochlea and had caused ankylosis of the stapes on each side. The structure of this large area was not uniform; however, the newly formed bone was exclusively of the weblike type and had an enormous amount of osteocytes; it had no interglobular spaces. At the mesial wall of the utricle and in the promontorium the new, weblike bone formed compact bone, with a great number of blood vessel channels. These channels ran in different directions and occasionally caused a starlike formation. They had a thick, hematoxylin-blue sheath and were largely obliterated; occasionally they contained a capillary. Where the newly formed, solid bone came in contact with the endosteum of the internal ear, it was atrophic and showed Howship lacunas, but practically no osteoclasts. (This is a typical finding in otosclerosis.) In no place did the new bone invade the internal ear. Not infrequently the newly formed, weblike bone included remnants of the original endochondral layer, consisting of lamellar bone (fig. 3).

At other sites in the otosclerotic area, there were marrow spaces of different sizes. In the frame of the oval window the marrow spaces were small, contained hyperemic blood vessels and connective tissue and were rich in cells (fig. 3). In these spaces there was considerable osteogenesis, but no bone resorption. At other sites the marrow spaces were large and may have extended to the macula cribrosa of the lateral or posterior semicircular canal, to the endosteum of the elliptic recess or to the periosteum of the cisterna perilymphatica. The content of the large marrow spaces differed; however, they invariably contained loose connective tissue, and they often contained hyperemic capillaries. Not infrequently small pieces of the original endochondral layer were embedded in the connective tissue (fig. 2 *2*). These pieces usually showed Howship lacunas; but there were no osteoclasts, and

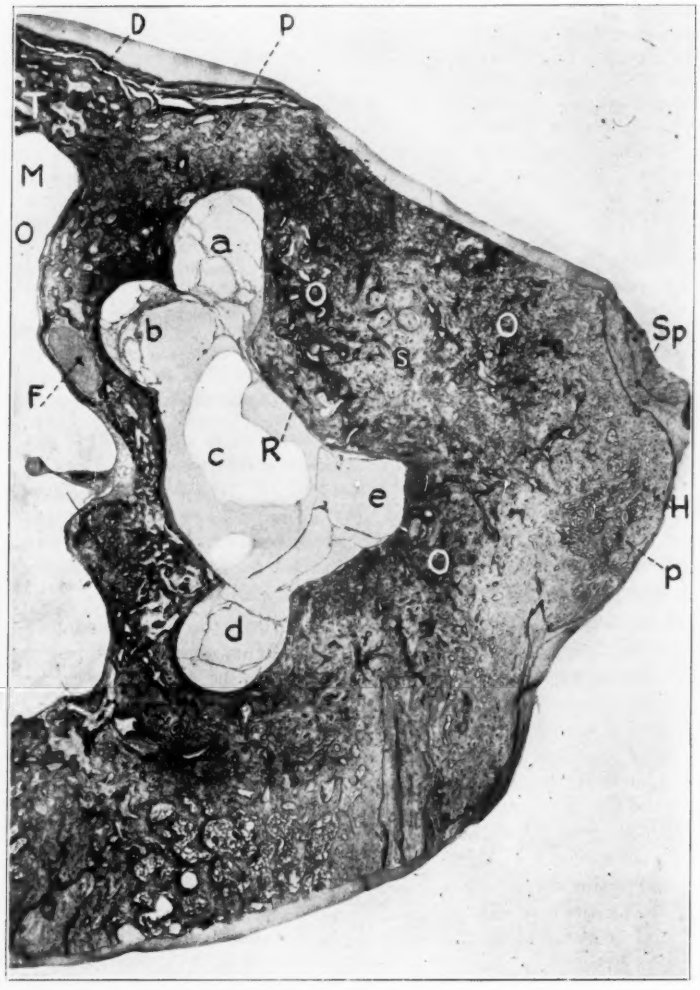

Fig. 3.—Section through the tympanic cavity (*M*), the superior ampulla (*a*), the lateral ampulla (*b*), the recessus ellipticus vestibuli (*c*), the inferior ampulla (*d*) and the crus commune (*e*); (*F*) facial nerve; (*T*) tegmen tympani; (*D*) dura; (*P*) periosteal layer of labyrinthine capsule; (*R*) remnant of original endochondral bone; (*O*) otosclerosis; (*s*) subarcuate fossa; (*Sp*) petrosal superior sinus; (*H*) hyperostosis.

the surface of the pieces was usually covered by a line of hematoxylin-blue cement. In addition, there was a metaplastic ossification within the connective tissue; however, the amount of newly formed bone was small (fig. 22). The boundaries of the large marrow spaces were aplastic and were covered by a hematoxylin-blue line. The otosclerotic area had sharp boundaries toward the remnants of the endochondral layer. Toward the periosteal layer the boundaries were not so sharp, but there was usually a line of separation.

Toward the ampullae of the semicircular canals, the otosclerotic area increased further in size and replaced, more and more, the periosteal layer above the superior semicircular canal. At this site the otosclerotic area extended to the dura of the middle cranial fossa, while there was a periosteal layer of extreme thickness and osteoporotic structure between the otosclerotic area and the dura of the posterior fossa. The osteoporotic periosteal layer adjacent to the dura of the posterior fossa bulged into the posterior fossa, forming the hyperostoses (fig. 3) mentioned in the foregoing report on the macroscopic examination of the temporal bones. It is interesting that the otosclerotic bone did not extend to the blood vessels and connective tissue of the subarcuate fossa (fig. 3). The latter was surrounded by the original bone of the capsule of the labyrinth. Toward the semicircular canals the otosclerotic area retained its extension; viz., otosclerotic bone embraced all semicircular canals, but again spared the area of the subarcuate fossa. It reached the dura of the middle fossa but not the dura of the posterior fossa. The structure of the bone in this area was essentially the same as in the area of the utricle.

COMMENT

The outstanding features of the present case were (1) otosclerosis, (2) osteoporosis, (3) hyperostoses at the posterior surface of the petrous bone and (4) labyrinthitis chronica ossificans.

Otosclerosis.—The changes supposed to be caused by otosclerosis occupied a huge area of the petrous bone. This area was greatest in the osseous capsule of the utricle (fig. 3) and in the semicircular canals. At this site the pathologic bone replaced almost the entire endochondral layer and a part of the periosteal layer; it extended to the dura of the middle fossa and toward the mucosa of the tympanic cavity. However, it spared the area of the subarcuate fossa and did not reach the dura of the posterior cranial fossa. Between the latter and the pathologic bone there was a periosteal layer, which, owing to the excess formation of pathologic bone within the petrous bone, bulged toward the posterior fossa, causing hyperostoses (fig. 3). This indicates that the hyperostoses of the present case must not be confounded with the exostoses frequently found in otosclerosis. The latter are actual outgrowths of the capsule of the labyrinth, while the hyperostoses of the present case were caused by an increase in the diameter of the petrous bone, which, in turn, was caused by excess formation of pathologic bone in the core of the pyramid.

Toward the saccule and the cochlea, the area of pathologic bone became smaller, while the area of osteoporosis correspondingly increased in size. The pathologic bone withdrew from the dura of the

middle fossa and from the mucosa of the tympanic cavity; in the area of the cochlea it came to an end, at the fundus of the internal auditory meatus (fig. *2 1*).

In this vast area, the bone of the original labyrinth capsule had been absorbed and replaced by a newly formed bone, as evidenced by the following features:

1. The bone had a weblike structure; in the normal endochondral layer of the mature labyrinth capsule there is no weblike bone.

2. The weblike bone did not contain interglobular spaces, consisting of cartilage cells; the normal endochondral layer does.

3. The weblike bone was formed in excess, causing hyperostoses. For this reason, the nourishment of the newly formed bone was insufficient, inasmuch as the blood vessels of the bone were largely obliterated. Consequently, the bone showed symptoms of decay, in particular close to the spaces of the internal ear. This is a characteristic feature of otosclerotic bone.[3] In contradistinction, the bone of the normal endochondral layer usually shows signs of senescence, indicated by the absence of osteocytes, but it does not show signs of decay.

4. The pathologic bone included fragments of the original endochondral layer (fig. *2 1* and fig. 3). The margins of these fragments frequently showed Howship lacunas, caused by the activity of osteoclasts, although osteoclasts were practically absent.

5. The pathologic bone included large marrow spaces, filled with connective tissue (fig. *2 2*); the normal endochondral layer in adults never contains large marrow spaces.

6. Within the large marrow spaces, frequent fragments of the original endochondral bone were noted (fig. *2 2*).

7. Within the marrow spaces, osteogenesis had frequently taken place (fig. *2 2*); in the normal endochondral layer in adults, there is no osteogenesis.

8. At the sites where the pathologic bone did not reach the maximum of extension, there was a sharp line between the pathologic and the normal bone.

Based on these data, the following concept of the development of the pathologic changes is most likely: A great part of the capsule of the labyrinth had been absorbed. All three layers of the capsule of the labyrinth, particularly the endochondral layer, were involved. The absorption did not involve the entire capsule: Fragments of the endochondral layer and several interglobular spaces had been spared. At the time of the death of the patient, the bone absorption, in general,

3. Brunner, H.: Beiträge zur Histogenese des otosklerotischen Knochens, Ztschr. f. Hals-, Nasen- u. Ohrenh. **6**:320, 1923.

had come to an end. The absorbed bone had been replaced by weblike bone, which usually forms by metaplastic ossification of connective tissue. In the area of the vestibule and the semicircular canals there was an excess formation of weblike bone; nevertheless, the newly formed bone had not entered the spaces of the internal ear, although it caused hyperostoses of the petrous bone. Osteogenesis took place at the time of the death of the patient; osteoclasis had ceased. These observations, in the absence of an infection of the tympanic cavity or the internal ear, indicated the diagnosis of a diffuse otosclerosis, which is not so frequent as the localized form of otosclerosis and which must not be confused with osteoporosis.

Otosclerosis and Osteoporosis.—The term "osteoporosis" indicates in general pathology an absorption of bone which slightly exceeds the limits of physiologic absorption, but after which there occurs no compensating deposition of newly formed bone. This causes the formation of large marrow spaces within the bone and renders the bone porous and brittle. In the temporal bones of senile persons this type of osteoporosis is not infrequent. In them, it concerns the external portion of the periosteal layer of the capsule of the labyrinth, where, under physiologic conditions, bone absorption and bone deposition continue up to old age, though in slight degree. Rarely, osteoporosis of the temporal bones extends into the endochondral layer,[4] where neither bone absorption nor deposition normally takes place in adult life. In these instances, the solid bone of the endochondral layer is transformed into cancellous bone. The new marrow spaces within the endochondral layer may contain connective tissue or fat, may extend to the endosteum and may cause an increase in the size of the involved pyramid. Although this patient reached only the age of 41 years, osteoporosis was noted in the area of the annulus tympanicus and in the ossicles (fig. 1) and in the osseous capsule of the cochlea (fig. 2). In the latter the condition involved the periosteal layer as well as the endochondral layer.

In the present case there was a reciprocal relationship between otosclerosis and osteoporosis. Where otosclerosis extended over a large area of the petrous bone osteoporosis became less pronounced, and vice versa. However, since the temporal bones of the patient were involved by both diseases, the question arises whether the changes caused by otosclerosis show deviations from the typical pattern which eventually may be caused by osteoporosis. In order to answer this difficult question, one must bear in mind that the changes in osteo-

4. (*a*) Wittmaack, K.: Ueber einen Befund von wahrer Spongiosierung der Labyrinthkapsel, Ztschr. f. Ohrenh. **77**:201, 1918. (*b*) Mayer.[1] (*c*) Nager, F. R., and Meyer, M.: Die Erkrankungen des Knochensystems und ihre Erscheinungen an der Innenohrkapsel des Menschen, Berlin, S. Karger, 1932.

porosis are primarily caused by a diminished activity of osteoblasts, while osteoclasts manifest a normal, or even slightly increased, activity. The diminished activity of the osteoblasts is supposed to be caused by diminished vitality of the person involved. The information on the otosclerotic area in this patient apparently indicates a precisely reversed activity of osteoblasts and osteoclasts; viz., an increased activity of osteoblasts and almost quiescent osteoclasts. In fact, there was a definite osteogenesis in the otosclerotic area even at the time of the patient's death. This apparently indicates that osteoporosis does not exert an influence on the progress of otosclerosis. Though Mayer [1] arrived at the same conclusion, it is not entirely correct. The diminished activity of the osteoblasts, which is the most characteristic feature of osteoporosis, concerns their ability to form lamellar bone, but not to form weblike bone. In the present case there was osteogenesis in the otosclerotic area, but the new bone was weblike, while lamellar bone was entirely absent. On the contrary, there was lamellar bone, though not in great amount, in the cases of diffuse otosclerosis studied by Nager and Meyer.[4c] These cases, however, were not associated with osteoporosis. Thus one arrives at the following conclusion: Osteoporosis apparently does not exert a strong influence on the progress of otosclerosis, but it may delay the formation of lamellar bone. The formation of weblike bone, caused by otosclerosis, is not counteracted by an osteoporosis of the capsule of the labyrinth.

Labyrinthitis Chronica Ossificans.—Labyrinthitis chronica ossificans is characterized by the formation of connective tissue and bone within the internal ear, particularly in the perilymphatic spaces, in the absence of inflammatory changes in the capsule of the labyrinth. Labyrinthitis chronica ossificans is usually the final result of a serous inflammation of the internal ear, the serous exudate being transformed into connective tissue and bone. Purulent inflammations of the internal ear may cause a similar condition; however, in these instances labyrinthitis chronica ossificans is associated with purulent inflammation of the capsule of the labyrinth or with scars after purulent inflammation. Labyrinthitis chronica ossificans occurs in syphilis, typhoid and leukemia and after head injuries. In the present case, it was associated with diffuse otosclerosis and osteoporosis. Likewise, Nager and Meyer [4c] noted labyrinthitis chronica ossificans in a few cases of diffuse otosclerosis.

This finding raises the question whether labyrinthitis chronica ossificans is caused by otosclerosis and osteoporosis of the capsule of the labyrinth. Many years ago Siebenmann [5] advanced the hypothesis that

5. Siebenmann, F.: Totaler knöcherner Verschluss beider Labyrinthfenster und Labyrinthitis serosa infolge progressiver Spongiosierung, Verhandl. d. deutsch. otol. Gesellsch. **20**:267, 1911.

in otosclerosis metabolites liberated by the pathologic changes of the bone may pass the endosteum of the internal ear and cause a serous labyrinthitis. Several years later, I made use of this hypothesis in order to explain the serous exudate which I had noticed in several cases of osteitis deformans of the capsule of the labyrinth.[6] If this hypothesis were correct, it would be logical to establish a relationship not only between otosclerosis and serous labyrinthitis, but likewise between otosclerosis and the final result of serous labyrinthitis, viz., labyrinthitis chronica ossificans. Nevertheless, this concept is by no means incontestable. It was acceptable as long as the known causes of serous labyrinthitis were (1) inflammations of the tympanic cavity or of the meninges, (2) systemic infections, (3) leukemia or (4) injuries to the head. If these causative factors were absent from a case of otosclerosis or from one of osteitis deformans associated with serous labyrinthitis, one was compelled to assume that the serous labyrinthitis was caused by the pathologic changes of the capsule of the labyrinth. At present, the problem is more complex, since we know that serous labyrinthitis may be caused not only by the factors mentioned but also by vasomotor disorders independent of these factors. Consequently it is not permissible to establish, in the present case, a relationship between otosclerosis and labyrinthitis chronica ossificans. What the cause of the labyrinthitis ossificans was in the present case cannot be determined, as a dependable history was not available. However, the fact that there have been many cases of labyrinthitis chronica ossificans without otosclerosis and a great number of instances of diffuse otosclerosis without labyrinthitis ossificans indicates that the labyrinthitis chronica ossificans of the present case must be considered purely coincidental.

The labyrinthitis chronica ossificans explained this patient's deafness. The deafness in these instances is always profound, usually complete. It causes deaf-mutism if the labyrinthitis ossificans occurred in childhood.[3a] It must, therefore, be borne in mind that deafness in otosclerosis is not invariably caused by the disease; it may be caused by a disease of the internal ear, coincidental to otosclerosis.

CONCLUSIONS

1. Hyperostoses of the petrous bone in diffuse otosclerosis may be caused by an increase in the diameter of the petrous bone, which, in turn, is caused by excess formation of pathologic bone in the core of the pyramid.

6. Brunner, H.: Ueber einen Fall von Pagetscher Krankheit, Monatschr. f. Ohrenh. **56**:810, 1922.

2. Osteoporosis apparently does not exert much influence on the progress of otosclerosis but it may delay the formation of lamellar bone. The formation of weblike bone, even in excess, caused by otosclerosis, is not counteracted by an osteoporosis of the capsule of the labyrinth.

3. Labyrinthitis chronica ossificans must be considered as purely coincidental to otosclerosis.

4. Deafness in otosclerosis is not necessarily caused by otosclerosis; it may be caused by a disease of the internal ear coincidental to otosclerosis, e. g., labyrinthitis chronica ossificans.

670 Clinton Avenue.

UNUSUAL OTITIC FOREIGN BODIES

Report of Two Cases

WILLIAM C. WARREN Jr., M.D.
AND
THOMAS E. BIVINS, M.D.
ATLANTA, GA.

A REVIEW of the literature in English since 1920 reveals reports of several unusual and interesting cases of a foreign body in the ear. Historically, each is an individual case, but all were the results of accidents. The purposes of this paper are: (1) to record 2 additional cases which are similar both in nature and in subsequent diagnosis and treatment, and (2) to indicate a procedure for reducing the recurrence of otitic disturbances in such cases. Necessity for examination with instruments is emphasized.

HISTORICAL REVIEW

Whale [1] reported an interesting otitic injury incurred by a soldier during World War I. A piece of shrapnel had been driven through both maxillary sinuses, the nasal cavity and the right orbit, with resulting destruction of the eye, and into the petrous bone. The soldier underwent several operations for plastic repair of the face and was considered cured. Approximately five years later he had a sudden onset of acute labyrinthitis, and roentgenograms revealed the metallic foreign body. A petromastoid operation effected removal of the shrapnel from the petrous bone, just above the superior semicircular canal.

Faulder [2] reported the case of a bead in the antrum tympanicum (mastoid antrum). He was unable to determine how the bead had gotten in the ear, or how long it had remained there. A simple mastoidectomy effected its removal, and the patient recovered without significant incident.

Three cases of lead bullets in the mastoid were reported by Hempstead. [3] The bullet in the first case caused intermittent otitis media

From the Department of Otorhinolaryngology, Emory University School of Medicine.

1. Whale, H. L.: An Unusual Case of Injury to the Petrous Bone, Lancet **2**:1002 (Nov. 12) 1921.

2. Faulder, T. J.: Foreign Body in the Mastoid Antrum, Proc. Roy. Soc. Med. **14**:26 (Nov.) 1920.

3. Hempstead, B. E.: Bullets in the Ear, J. A. M. A. **98**:2281 (June 25) 1932.

for a number of years, but it was not discovered and removed until sixteen years after its entry. In the second case, the bullet remained in the ear for eight years before its removal, by radical mastoidectomy. In Hempstead's last case, a bullet remained in the middle ear for five years before symptoms required its removal.

Bettington [4] reported the case of a 5 year old boy with a pussy willow catkin in his ear. Part of the foreign body had been removed at the time of the injury, and more had been removed later, with the use of general anesthesia. The ear did not heal, and six months later a simple mastoidectomy revealed the rest of the foreign body to be in the aditus.

Injuries of long duration in the ear were also reported by Mosher.[5] In 1 case a wound, made by a knife blade, through the external auditory canal into the middle cranial fossa caused meningitis in seventy-two hours. The knife blade was removed by mastoidectomy, and the tip of the blade was found just above the jugular bulb, in the middle cranial fossa. Mosher's second case was that of a 60 year old man who had been shot in the left mastoid. The ear gave him little trouble for thirteen years, after which signs of purulent otitis media developed. Fifteen lead fragments were removed from the mastoid region. During the operation an extradural abscess was discovered and drained. A radical mastoidectomy was eventually required to effect recovery.

In 1 of Wood's patients, a part of a scissors blade was found in the medial wall of the middle ear.[6] A raised window had fallen on a 4 year old child. The immediate result was complete facial paralysis; a few days later purulent otitis media developed. An otologist was consulted about nine months later, and roentgenograms revealed a metallic foreign body in the ear. At first it was thought to be leaded glass from the window, but radical operation revealed part of a scissors blade in the medial wall of the middle ear. The scissors, the family recalled, had been left in the window prior to the child's injury.

REPORT OF CASES

Case 1.—A Negro, 66 years of age, was referred to the otology service of Grady Memorial Hospital on Dec. 14, 1946, with a diagnosis of purulent otitis media of the left ear. For about two weeks prior to his admission to the hospital the patient had been treated locally, with sulfadiazine administered orally and with injections of penicillin. An accurate history was not obtainable because of the patient's faulty memory, but he stated the belief that about four years

4. Bettington, R. H.: Peculiar Sequel to a Foreign Body in the Ear, M. J. Australia **1**:535 (March 19) 1938.

5. Mosher, W. F.: Foreign Bodies in the External Canal, Middle Ear and Mastoid and Their Complications, Arch. Otolaryng. **36**:679 (Nov.) 1942.

6. Wood, V. V.: Scissors Point Embedded in the Inner Tympanic Wall, with Facial Paralysis, Ann. Otol., Rhin. & Laryng. **39**:111 (March) 1930.

prior to the examination his left ear had discharged pus for two months. He had had no medical treatment at the time; the ear stopped draining spontaneously.

Ten months prior to his admission pus began draining profusely from his ear; during the next months he had what he described as "black-out spells" but he denied having experienced vertigo, fainting or unconsciousness. His health in general had been good. Poor vision, especially in the left eye, was his only complaint other than the discharge from his ear. He failed to comment on his poor hearing on the left side until asked about it. Then he said that he believed the ear was a little deaf but that he did not know how long such a condition had existed.

Physical Examination.—The patient was well developed and adequately nourished and looked his age. He was rational and oriented and seemed to hear well. He had complete paralysis of the left side of the face, and the left eye did not move laterally when he looked to the left. His vision of 60/100 in each eye was considered good for distance. The eyegrounds showed nothing significant except arteriosclerotic retinopathy grade 1. His few remaining teeth were carious, with a 4 plus degree of pyorrhea. The right ear was normal;

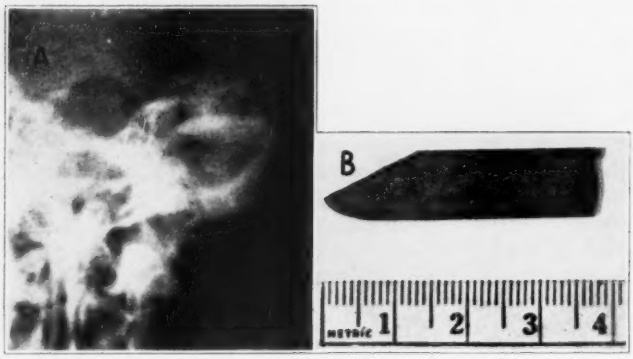

Fig. 1.—*A*, roentgenogram indicating foreign body in superior aspect of left petrous bone; *B*, knife blade after removal.

hearing in that ear was approximately normal. The left ear had a profuse, foul, purulent discharge. Since the external canal was filled with a polyp-like growth, the drum membrance could not be seen. When the polyp was removed, neither the tympanic membrane nor the ossicles could be seen. No tenderness was elicited over the mastoid region. Hearing was then about 1/15 for a spoken voice; bone conduction was good. Complete neurologic examination revealed no abnormality except palsies in the sixth and seventh cerebral nerves. The blood pressure was 110 systolic and 70 diastolic. The patient's temperature was 98.0 F.

Laboratory Examination.—The white blood cell count was 6,500; the urine was normal and the Kahn reaction of the blood was negative. Examination of the spinal fluid was not accomplished.

Roentgenograms of the mastoid and petrous portions indicated an opaque foreign body, measuring 3.7 cm. in length, lying in the superior aspect of the left petrous bone (fig. 1*A*). The foreign body extended from the bony wall

of the external auditory canal to a point approximately 5 mm. inferior to the tip of the petrous bone. The entire petrous bone had the appearance of sclerosis consistent with petrositis. No cavities were noted in the petrous bone.

Operation.—On Dec. 31, 1946, surgical removal of the foreign body was accomplished by an endaural approach. The membranous wall of the external auditory canal was incised down to the annulus tympanicus, and the outer end of the foreign body was located in the anterior wall of the bony canal. It was easily removed with grasping forceps. The object was a rusty knife blade, 3.7 by 1 cc. (fig. 1*B*). When interrogated after the operation, the patient said that he had been stabbed in the ear about thirty years ago.

The exact cause of the facial and ocular palsies was not determined. The patient could not state the date on which they were first noticed, but he expressed the belief that the ocular paralysis was recent. When he was last seen, on

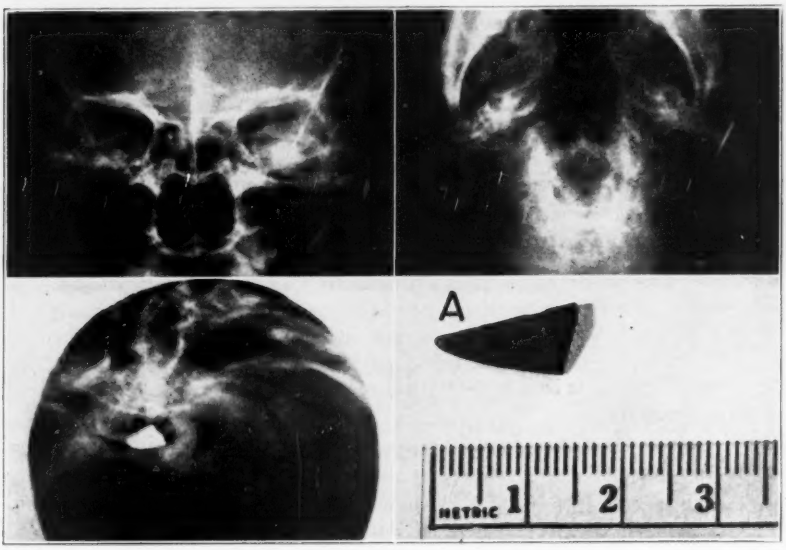

Fig. 2.—Roentgenograms indicating foreign body in region of left mastoid process. *A*, knife blade after removal.

March 7, 1947, the palsy of the sixth nerve had completely disappeared, but the facial palsy was unchanged. His ear had healed; no purulent discharge could be seen. His hearing was about 3/15 for whispered voice.

Case 2.—A 34 year old Negro woman was admitted to the Grady Memorial Hospital on Feb. 12, 1947. She stated that she had been in a knife fight about November 1937 and had received a stab wound in front of the left ear. The laceration was treated at that time in the outpatient service, but the patient knew nothing of a knife blade's being in her ear, and the intern did not probe for possible foreign bodies. She had no otologic complaints until November 1945, when a large abscess over the left postauricular region developed and ruptured into the external auditory canal. She received outpatient treatment in

the clinic, the discharge stopped, and for the next fifteen months she had no otologic complaints except fleeting pains in the left ear. One week prior to admission, she began to have pain, tenderness and swelling over the postauricular area. The symptoms progressed to the point of extreme tenderness and pain in the mastoid region. Roentgenograms were taken for the first time (fig. 2), and the following report was made:

"The films indicate a broken knife blade, measuring approximately 1.5 cm. in length, in the region of the left mastoid process. The broken end of the blade is in the region of the posterior inferior wall of the external auditory canal. The tip of the blade has apparently entered the mastoid process anterior to the stylomastoid foramen. The mastoid cells appear to be sclerotic, but there is no evidence of mastoiditis or cholesteatoma."

The patient's hearing was never affected. The knife blade was removed in a simple mastoidectomy, by the postauricular route (fig. 2). The mastoid cells were sclerotic. The patient had an uneventful recovery.

SUMMARY

Two unusual cases of foreign bodies in the ear are reported. In each case the origin was traumatic, and the foreign body was present for a number of years before a pathologic process in the ears was manifest to the patient or to the physician.

CONCLUSION

From the review of the literature and the present cases, it can be said that prolonged purulent otitis media may be caused by foreign bodies, of whose presence the patient is not necessarily aware. In all suspicious cases, therefore, roentgenoscopic examination should be made to determine whether a foreign body is present. Such a procedure would contribute both to the welfare of the patient and to the protection of the physician.

Emory University School of Medicine.

p_H OF THE CUTANEOUS SURFACE OF THE EXTERNAL AUDITORY CANAL

A Study of Twenty-Seven Infants, Forty-Four Children and Sixty Adults

NOAH D. FABRICANT, M.D.
AND
M. A. PERLSTEIN, M.D.
CHICAGO

ALTHOUGH it has been variously estimated that otitis externa constitutes from 5 to 40 per cent of all cases encountered in otologic practice in the United States [1]—the wide variation being due to sectional differences in temperature and humidity—renewed interest in infection of the external auditory canal is essentially a by-product of the war years. This situation is the result of the high incidence and widespread distribution of otitis externa among military personnel in humid tropical and subtropical regions. While the causative agent of otitis externa can be any one of a variety of bacteria and fungi, it is quite probable that the role of the latter, whether as primary or secondary agents of infection, has been exaggerated.[2]

The normal human skin harbors an enormous number of bacteria, which ordinarily are harmless. According to Price,[3] these may be divided into a resident and a transient population, the former usually remaining fairly stable. However, some influences, particularly heat and moisture, cause considerable changes in the number of bacteria found on normal skin. Sometimes pathogenic bacteria suddenly become members of the resident flora of the skin; they may then be extremely persistent and difficult to dislodge. Once a foothold is gained in skin already diseased, bacteria are responsible, in greater or less degree, for many diseases affecting the human skin. By virtue of its exposed position to the outside world, the external auditory canal is open to invasion by numerous micro-organisms; here a moist milieu containing cellular detritus and cerumen often serves as an adequate culture medium for the growth of bacteria and fungi.

1. Gill, W. D.: Otitis Externa, Ann. Otol., Rhin. & Laryng. **51**:370 (June) 1942.

2. Syverton, J. T.; Hess, W. R., and Krafchuk, J.: Otitis Externa: Clinical Observations and Microbiologic Flora, Arch. Otolaryng. **43**:213 (March) 1946.

3. Price, P. B.: The Bacteriology of the Normal Skin: A New Quantitative Test Applied to a Study of the Bacterial Flora and the Disinfectant Action of Mechanical Cleansing, J. Infect. Dis. **63**:301 (Nov.-Dec.) 1938.

In the opinion expressed by Pillsbury,[4] the problem of the management of bacterial infections of the skin cannot be dismissed simply by a discussion of the diseases produced, the bacteria concerned and the disadvantages of the various antibacterial agents now available, especially since these agents still have certain definite shortcomings. The management of the more difficult and more resistant cutaneous infections can be improved by a better understanding of the manner in which the skin resists and harbors bacteria and of some of the mechanisms by which bacteria produce harmful effects. Several factors apparently play a role in the remarkable protection afforded by the normal human skin against bacterial invasion. These factors, however, are imperfectly understood, and further basic study is necessary.

A factor of considerable importance is the p_H of the skin. Marchionini and Hausknecht[5] determined the p_H of the skin and found that the surface of the skin is enveloped (figuratively speaking) by an "acid cloak," which exhibits pronounced defense against bacterial invasion. The normal "acid mantle" of the skin's surface is dependent on cornified horny cells, whose reaction is more acid than that of any other cutaneous tissue, sweat, secretions of the sebaceous or oil glands, and the apocrine glands, which are probably the chief producers of the odoriferous cutaneous secretions.

In a series of studies, Blank[6] measured the p_H of the surface of the skin of a series of 100 male medical students and 100 women nurses, aged 19 to 27, and found the p_H to vary from 4.0 to 7.0. Most of the p_H readings were found to lie between 4.2 and 5.6, the average p_H of female subjects being 0.5 higher than that of male subjects. Examination of children, aged 3 to 14 years, revealed the variation in p_H to be the same as that in adults, namely, 4.0 to 7.0. Most of the p_H readings fell between 4.2 and 6.0, the average p_H of girls being from 0.2 to 0.3 higher than that of boys.

In investigating the p_H of the skin of seborrheic patients, Marchionini found that the seborrheic areas of the body are more alkaline than other parts of the skin's surface. The alkaline areas were shown to be less bactericidal than the rest of the skin, thus being predisposed

4. Pillsbury, D. M.: The Management of Bacterial Infections of the Skin, J. A. M. A. **132**:692 (Nov. 23) 1946.

5. Marchionini, A., and Hausknecht, W.: Säuremantel der Haut und Bakterienabwehr: I. Die regionäre Verschiedenheit der Wasserstoffionenkonzentration der Hautoberfläche, Klin. Wchnschr. **17**:663 (May 7) 1938; II. Ueber die regionäre Verschiedenheit der Bakterienabwehr und Desinfektionskraft der Hautoberfläche, ibid. **17**:736 (May 21) 1938.

6. Blank, I. H.: Measurement of p_H of the Skin Surface: I. Technique, J. Invest. Dermat. **2**:67 (April) 1939; III. Measurements on the Arms of Children with no Apparent Skin Lesions, ibid. **2**:231 (Oct.) 1939.

to cutaneous infection. Marchionini also demonstrated that there is a shift of the p_H of the skin toward the alkaline side in most cases of vesicular eczema. Some dermatoses are associated with excessive dryness of the skin; as the dryness becomes more pronounced, the tendency to fissure formation is increased; this tendency, in turn, contributes to an increase in alkalinity. Bernstein and Herrmann,[7] in studies of the p_H of the skin surface, found the highest degree of alkalinity in fissures present in eczematous processes.

Susceptibility to infection by bacteria is thought by many investigators to be due to the removal of the antibacterial "acid mantle" of the skin and its displacement by an abnormally "alkaline mantle." As in the case of bacterial micro-organisms, warmth and moisture are especially favorable to the propagation of many forms of fungi; the commonest fungi are known to multiply in the fertile soil supplied by damaged and swollen horny material. Indeed, moisture, maceration, dead horny tissue and relative alkalinity are four favorite conditions for the propagation of fungi causing such conditions as moniliasis, dermatophytosis ("athlete's foot") and ringworm of the groin.

In commenting on the acidity of the skin, Wise and Sulzberger[8] declared:

> These results confirm the fact of the normal acidity of the skin surface and the old-accepted concept of the protective function of the acid mantle. The logical conclusion, in managing many skin diseases is, therefore, not (as so popularly and inanely reiterated) to "alkalinize" but rather to "acidify"—at least as regards the skin's surface. Experimental data show not that there is "too much acid" but rather that there may be too little acid on the skin surfaces in many instances of cutaneous infection and disease.

Generally, clinical experience has shown that alkalis are much more likely to produce lasting or severe cutaneous damage than are acids.

PRESENT INVESTIGATION

It will be recalled that the external auditory canal consists of a membranocartilaginous and a bony part, the entirety measuring about 1¼ inches (3.2 cm.) in length. The membranocartilaginous portion constitutes the outer one third of the canal, while the medial two thirds is bony. Development of the canal is complete by the third year of life. Hair follicles and sebaceous and ceruminous glands are present only in the membranocartilaginous portion, being absent in the bony part. It is the outer one third of the canal which is the seat of furuncles.

7. Bernstein, E. T., and Herrmann, F.: The Acidity on the Surface of the Skin, New York State J. Med. **42**:436 (March 1) 1942.

8. Wise, F., and Sulzberger, M. B.: Year Book of Dermatology and Syphilology, Chicago, The Year Book Publishers, Inc., 1938, p. 553.

TABLE 1.—*Data on the p_H of the Cutaneous Surface of the Normal External Auditory Canal in 60 Adults*

Patient	Age	Sex	Normal p_H of Skin	Patient	Age	Sex	Normal p_H of Skin
1	42	M	6.5 6.7 6.9	24	36	F	5.0 5.2 5.2
2	33	F	6.4 6.7 6.7	25	25	F	7.1 6.9 6.8
3	36	M	6.8 6.8 6.8	26	30	M	6.7 6.9 6.9
4	37	F	6.8 7.0 6.9	27	31	M	7.8 7.7 7.7
5	47	M	6.4 6.1 5.9	28	27	F	6.9 7.0 7.1
6	36	M	6.4 6.6 6.6	29	45	F	6.4 6.5 6.2
7	34	M	7.0 6.9 7.0	30	37	M	7.0 7.0 7.0
8	33	F	7.0 7.0 6.9	31	43	F	7.0 6.9 6.9
9	34	M	6.0 6.3 6.4	32	42	M	6.9 6.8 6.8
10	41	F	6.5 6.7 6.6	33	29	M	5.2 5.0 5.0
11	46	M	6.6 6.5 6.5	34	32	M	6.8 6.8 6.8
12	31	M	7.0 7.1 7.0	35	31	M	6.6 6.9 6.9
13	34	F	6.7 6.9 7.0	36	26	F	6.9 7.0 7.1
14	28	M	6.5 6.7 6.7	37	22	M	6.6 6.7 6.8
15	26	F	7.3 7.3 7.6	38	36	M	7.1 7.5 7.7
16	37	F	7.3 7.3 7.3	39	25	F	7.4 7.4 7.4
17	35	M	7.3 7.3 7.2	40	21	F	7.6 7.5 7.3
18	39	F	6.5 6.7 6.9	41	21	M	7.2 7.1 7.1
19	28	F	6.7 6.7 6.7	42	31	M	5.8 5.8 5.8
20	29	F	6.8 6.8 6.9	43	29	F	6.4 6.4 6.6
21	42	M	5.3 5.0 5.0	44	29	F	6.5 6.5 6.7
22	35	M	7.6 7.7 7.7	45	33	M	6.6 6.5 6.5
23	34	F	6.0 5.9 5.8	46	43	M	6.5 6.7 6.7

TABLE 1.—*Data on the p_H of the Cutaneous Surface of the Normal External Auditory Canal in 60 Adults—Continued*

Patient	Age	Sex	Normal p_H of Skin	Patient	Age	Sex	Normal p_H of Skin
47	27	F	7.1 7.2 7.3	54	26	F	6.7 6.9 7.0
48	37	M	6.2 6.2 6.4	55	29	M	6.0 6.0 6.0
49	39	F	6.7 6.5 6.8	56	23	F	7.0 7.0 6.9
50	46	M	6.3 6.5 6.5	57	44	F	6.3 6.9 7.0
51	39	F	6.9 6.8 6.9	58	46	M	6.3 6.5 6.6
52	30	F	6.4 6.6 6.5	59	39	F	5.9 6.2 6.2
53	32	M	6.8 6.7 6.9	60	44	F	7.0 7.2 7.4

In the present investigation, measurement of the p_H of the cutaneous surface of the external auditory canal was undertaken in a series of 131 subjects (27 infants, 44 children and 60 adults) with no apparent lesions in their external auditory canals and either with no visible cerumen or with minimal amounts. A specially adapted silver–silver chloride glass electrode, employed in conjunction with the Coleman electrometer, was introduced into the external auditory canal so as to assure adequate contact with the cutaneous surface of the membrano-cartilaginous portion. Because of the inherent difficulty of carrying on p_H readings of the skin of the external auditory canal in infants and very young children over prolonged periods, for the sake of uniformity three consecutive readings were taken at one-half minute intervals in all three groups—infants, children and adults.

The 60 adults under study (30 men and 30 women) were parents of the infants and children. The ages of the men ranged from 21 to 47; the ages of the women, from 21 to 45. In the adult male group the p_H range of the skin of the external auditory canal was found to be from 5.0 to 7.8. In the adult female group the p_H range of the skin of the external auditory canal was found to be from 5.0 to 7.6. Of the 30 adult subjects, 23 evinced p_H values for the skin entirely within the acid range; 6, values within the alkaline range, and 1, a neutral p_H. Of the 30 adult female subjects, 21 evinced p_H values for the skin within the acid range, 6 within the alkaline range and 3 within a slightly acid, slightly alkaline range of fluctuations (table 1). From these observations, it appears that there is little difference in the p_H range of the skin of the normal external auditory canal of men and women of similar age groups. The p_H values fall chiefly within the acid range.

TABLE 2.—*Data on the p_H of the Cutaneous Surface of the Normal External Auditory Canal in 71 Infants and Children*

Patient	Age	Sex	Normal p_H of Skin	Patient	Age	Sex	Normal p_H of Skin
1	2½	F	6.7 7.0 7.2	24	5	M	6.0 6.0 6.2
2	9 mo.	M	7.5 7.5 7.6	25	8½	F	5.9 5.8 5.7
3	4½	M	7.1 7.3 7.4	26	2	F	6.2 6.3 6.3
4	11 mo.	F	6.1 6.6 6.7	27	2	M	6.2 6.7 6.6
5	2	M	6.9 7.0 7.0	28	18 mo.	F	6.0 6.3 6.4
6	3	M	7.1 7.1 7.2	29	2½	F	6.5 6.7 6.7
7	3 mo.	M	5.7 6.2 6.2	30	5	F	6.7 6.9 6.9
8	4	F	7.1 7.5 7.7	31	7	F	6.2 6.9 6.9
9	9½	M	7.0 7.0 7.1	32	5	M	7.0 7.4 7.5
10	12	M	7.5 7.6 7.7	33	12	F	7.8 7.9 7.8
11	18 mo.	M	6.5 6.6 7.0	34	9	F	6.9 7.0 6.9
12	2½	M	7.1 7.4 7.6	35	4	M	7.9 7.9 7.8
13	5½	M	7.7 7.4 7.7	36	1	M	7.5 7.4 7.6
14	3	M	6.4 6.4 6.4	37	4	M	6.3 6.7 6.7
15	8 mo.	M	6.1 6.3 6.5	38	15 mo.	F	7.4 7.4 7.4
16	11	M	7.5 7.5 7.4	39	3	M	6.5 6.1 6.5
17	13½ mo.	F	5.7 5.8 5.7	40	4 mo.	M	6.2 6.2 6.2
18	6	M	6.9 7.1 7.4	41	7½	M	6.7 6.9 7.0
19	13	F	6.8 6.9 7.0	42	10 mo.	F	6.0 6.3 6.3
20	10 wk.	F	6.2 6.1 6.3	43	23 mo.	F	5.6 6.2 5.6
21	2½	F	6.4 6.5 6.8	44	6 mo.	M	5.6 5.8 5.9
22	5	F	5.9 6.0 6.2	45	5 mo.	M	6.6 6.9 6.8
23	3	M	6.5 6.6 6.7	46	5 wk.	F	5.7 6.0 6.0

TABLE 2.—*Data on the p_H of the Cutaneous Surface of the Normal External Auditory Canal in 71 Infants and Children—Continued*

Patient	Age	Sex	Normal p_H of Skin	Patient	Age	Sex	Normal p_H of Skin
47	4	M	7.0 7.2 7.2	60	7	M	6.0 6.1 6.0
48	10	M	7.5 7.5 7.6	61	9	F	7.0 7.2 7.5
49	8 mo.	F	6.9 6.7 6.8	62	7	F	7.0 7.1 7.1
50	5	F	6.5 6.5 6.6	63	5	F	6.9 6.8 6.9
51	10½ mo.	F	6.6 6.5 6.7	64	17 mo.	M	6.7 6.8 6.9
52	4	M	6.4 6.5 6.6	65	14 mo.	M	6.0 6.2 6.1
53	6	M	7.2 7.3 7.5	66	14 mo.	F	6.2 6.3 6.3
54	8	F	6.5 6.8 6.9	67	4	M	7.4 7.3 7.3
55	2½	M	7.3 7.1 7.3	68	10	M	7.7 7.7 7.7
56	11	M	6.6 6.6 6.8	69	4 mo.	F	5.2 5.5 5.5
57	3	M	6.3 6.6 6.5	70	6	F	7.0 7.1 7.0
58	8 mo.	M	7.5 7.6 7.5	71	6½	F	6.8 6.9 6.9
59	17 mo.	M	6.1 6.0 6.2				

The younger subjects under investigation numbered 27 infants (14 males and 13 females) and 44 children (23 males and 21 females). The ages of the male infants ranged from 3 months to 2 years and those of female infants from 5 weeks to 2 years. The ages of the male children were from 2 to 11 years and those of the female children from 2 to 13 years. In the group of male infants, the p_H range of the skin of the external auditory canal was from 5.6 to 7.6. In the group of female infants, the p_H range of the skin of the external auditory canal was found to be from 5.2 to 7.4. Male children, on the other hand, had a p_H range of the skin from 6.0 to 7.9, whereas female children had a range from 5.7 to 7.9 (table 2).

Of the 14 male infants, 11 evinced p_H values for the skin within the acid range, while values for 3 subjects fell within the alkaline range. Of the 13 female infants, 12 evinced p_H values of the skin entirely within the acid range and but 1 within the alkaline range. Of the 23 male children, 10 evinced p_H values for the skin within the acid range,

12 within the alkaline range and 1 within a slightly acid, slightly alkaline range of fluctuations. Of the 21 female children, 12 evinced p_H values for the skin within the acid range, 8 within the alkaline range and 1 within a slightly acid, slightly alkaline range of fluctuations.

From these observations it appears that there is little difference in the p_H range of the skin of the normal external auditory canal of male infants and that of female infants. The p_H values fall mostly within the acid range. Among children, the tendency for acid p_H values is not nearly so pronounced as that for other groups.

SUMMARY AND CONCLUSIONS

Renewed interest in infection of the external auditory canal is essentially a by-product of the war years. By virtue of its exposed position to the outside world, the external auditory canal is open to invasion by numerous micro-organisms; a moist milieu containing cellular detritus and cerumen often serves as an adequate culture medium for the growth of bacteria and fungi.

The management of the more difficult and more resistant cutaneous infections can be improved by a better understanding of the manner in which the skin resists and harbors bacteria and of some of the mechanisms by which bacteria produce harmful effects. Several factors play a role in the remarkable protection afforded by the normal human skin against bacterial invasion. These factors, however, are imperfectly understood, and further basic study is necessary.

A factor of considerable importance is the p_H of the skin. The surface of the skin is enveloped by an "acid mantle," which exhibits pronounced defense against bacterial invasion. Determinations of the p_H of the skin by other investigators reveal the following facts: (1) The seborrheic areas of the body are more alkaline than other parts of the skin's surface; (2) the alkaline areas are less bactericidal than the rest of the skin, thus being predisposed to cutaneous infection; (3) in most cases of vesicular eczema there is a shift of the p_H of the skin toward the alkaline side; (4) the highest degree of alkalinity is present in fissures associated with eczematous processes, and (5) moisture, maceration, dead horny tissue and relative alkalinity are four favorite conditions for the propagation of many forms of fungi. It has been asserted:[8]

> The logical conclusion in managing many skin diseases, is not to "alkalinize" but rather to "acidify"—at least as regards the skin's surface.

In the present investigation, measurement of the p_H of the cutaneous surface of the external auditory canal was undertaken in a series of 131 subjects (27 infants, 44 children and 60 adults) with no apparent lesions in their external auditory canals and either with no visible

cerumen or with minimal amounts. A specially adapted glass electrode was employed in conjunction with the Coleman electrometer.

In the group of adult male subjects, the p_H range of the skin of the external auditory canal was found to be from 5.0 to 7.8. In the group of adult female subjects, the p_H range of the skin of the external auditory canal was found to be from 5.0 to 7.6. Of 30 adult male subjects, 23 evinced p_H values for the skin within the acid range; 6, values within the alkaline range, and 1, a neutral p_H. Of 30 women, 21 evinced p_H values for the skin within the acid range, 6 within the alkaline range and 3 within the slightly acid, slightly alkaline range of fluctuations. From these observations it appears that there is little difference in the p_H range of the skin of the normal external auditory canal of adult male and adult female subjects of similar age groups. The p_H values fall chiefly within the acid range.

In the group of male infants, the p_H range of the skin of the external auditory canal was found to be from 5.6 to 7.6. In the group of female infants the p_H range of the skin of the external auditory canal was found to be from 5.2 to 7.4. Male children, on the other hand, had a range in the p_H of the skin from 6.0 to 7.9, whereas female children had a range in the p_H of the skin of from 5.7 to 7.9. Of 14 male infants, 11 evinced p_H values for the skin within the acid range, while those for 3 subjects fell within the alkaline range. Of 13 female infants, 12 evinced p_H values for the skin within the acid range and but 1 within the alkaline range. Of 23 male children, 10 evinced p_H values for the skin within the acid range, 12 within the alkaline range and 1 within a slightly acid, slightly alkaline range of fluctuations. Of 21 female children, 12 evinced p_H values for the skin within the acid range, 8 within the alkaline range and 1 within a slightly acid, slightly alkaline range of fluctuations. From these observations it appears that there is little difference in the p_H range of the skin of the normal external auditory canal of male and of female infants. The p_H values fall chiefly within the acid range. Among children the tendency for acid p_H values is not nearly as distinct as it is for other groups.

185 North Wabash Avenue.

PROGRESS IN SURGERY OF THE SEPTUM

REUBEN KAYSER, M.D.
Assistant Surgeon Manhattan Eye, Ear and Throat Hospital
BROOKLYN

IN A previous presentation, it was demonstrated how with the new procedure [1] one could, with safety, entirely excise a deformed septal cartilage and still support the nose by the use of the upper lateral cartilages and a columellar cartilaginous strut. Since then various modifications of the procedure have been advanced by other investigators (Fomon and others [2]). One modification by Galloway [3] in my opinion destroys what I consider the shock absorber action of the membranous septum because it traverses the membranous septum with a rigid cartilage graft. A columellar strut implanted into the columella by the Fomon technic is a more sound procedure physiologically. My experience has taught me that it is futile to attempt to correct scar contractures of the mucoperichondrial flaps by the employment of septal cartilage grafts. Regardless of the type of septal deformity, its correction must be achieved along the lines of proved surgical and plastic principles applied by an otolaryngologist trained in rhinoplasty who can improvise as the occasion arises.

I have waited before publishing many of the findings reported in 1944 because I felt that it was necessary to take sufficient time to evaluate the merits of the contributions made. Since the original presentation in 1944 by Kayser of the studies to be mentioned in this paper, there have been no modifications of any of the observations then noted, and they are now presented as then reported. Any operator

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Part of paper read before the American Otorhinologic Society for the Advancement for Plastic and Reconstructive Surgery, Inc., under the title "Rhinoplasty and Nasal Rhysiology" on November 11, 1944. Also read before the Section on Laryngology, Rhinology and Otology of the Medical Society of the County of Kings and Academy of Medicine of Brooklyn, on April 11, 1945, as part of paper titled "Rhinoplasty and the Submucous Resection as Aids Towards Improving Nasal Physiology with Presentation of a New Technic for Correcting the Dislocated and Obstructing Septal Cartilage Associated With and Without External Nasal Deformity."

1. Kayser, R.: New Operation for Dislocated Septal Cartilage, Am. J. Surg. **72**:242-251 (Aug.) 1946.

2. Fomon, S.; Syracuse, V. R.; Bolotow, N., and Pullen, M.: Plastic Repair of the Deflected Nasal Septum, Arch. Otolaryng. **44**:141-156 (Aug.) 1946.

3. Galloway, cited by Formon, Syracuse, Bolotow and Pullen.[2]

who is familiar with the post-traumatic distortions of the septal cartilage and bone and the scarring and contractures of the surrounding soft parts knows that although the correction of each septal deformity poses an individual problem the basic principle of working close to the bone and cartilage is a sine qua non for a satisfactory submucous resection. Similarly, I am now convinced that the Kayser procedure has simplified surgery of the septum because of its pioneer work in establishing the principle that one can, in selected cases, remove the entire septal cartilage without the danger of a saddle deformity.

Proetz [4] said that "aside from the removal of tumors and the correction of cosmetic shortcomings, surgery of the nose has only two functions—to eradicate infection and to restore physiologic harmony." This paper is concerned with the restoration of physiologic harmony, to be achieved by a submucous resection which may necessitate for its complete execution rhinoplastic procedures on the nasal framework. I wish to establish the concept that such rhinoplastic procedures as the removal of a nasal hump, the out-fracturing incidental to a lateral osteotomy or the transfixion of the nasal tip can facilitate the thorough execution of a complicated submucous resection. However, the fact cannot be stressed too strongly that no rule of thumb can replace the judgment of an experienced otolaryngologist in deciding when these steps are to be employed.

It is now the teaching on the service of Dr. Anthony Nigro at the Manhattan Eye, Ear and Throat Hospital that a knowledge of various systems and subdivisions of procedures is of little value if one is not familiar with the following facts. First one must know the physiologic function of the nose. More important still is a thorough knowledge not only of all the normal variations to be found in the anatomy of the bones, cartilages and soft structures of the nose but also of the distortions of these parts caused by trauma.

The principle functions which the rhinologist must conserve are:

1. Sense of smell. Inasmuch as this is limited to the area of the distribution of the olfactory nerve, it is important that this area be kept clear.

2. Comfortable breathing of air currents. According to Proetz,[4] "both inspiratory and expiratory air currents pass in a high curve through the nasal chambers but only the expiratory currents enter the meatuses." It is evident that the direction of the nares as well as their size and shape can influence these airways. Nasal deformities which obstruct the high curve mentioned obstruct the airways. Any deformity which obstructs these airways must be corrected.

4. Proetz, A. W.: Essays on the Applied Physiology of the Nose, St. Louis, Annals Publishing Company, 1941, p. 349.

3. Moistening of the air. The nose secretes 1 liter of water every twenty-four hours. A protective blanket of mucus must be maintained on the nasal mucosa. The villi must be protected from drying. As deviated septum can cause areas of drying, with subsequent catarrh and even headaches, the correction of such a septum by a submucous resection is physiologically sound.

4. Heating of the air. Two and a half per cent of the loss of body heat occurs in the nose, where the air is heated to the body temperature. In order to achieve this the turbinates act as wall radiators. Because the outer framework of the nose is rigid, the turbinates by their capacity to swell quickly act as valves to the volume of air passing over them. Deformities of the nose and the septum accompanied with septal spurs and ridges which dig into the turbinates limit the capacity of the turbinates to respond physiologically to normal stimuli. Here too, therefore, the indicated operation is physiologically sound.

The following terminology will help clarify the description of the structures of the nose and their relationships to each other. Anterior and ventral are used interchangeably—away from the back. Posterior and dorsal are used interchangeably—toward the back. Cephalad means in the direction of the cranium; caudad, in the direction of the feet; ental, toward the center; ectal, in the direction of the surface; medial, toward the midline, and lateral, away from the midline. Thus the so-called dorsum of the nose is really the ventral or anterior aspect of the nose. The tip of the nose is at the anterior caudad end.

STRUCTURE OF THE NASAL FRAMEWORK

This discussion is concerned with the anatomy of the structures of the nasal framework and of the septum. The nasal framework is composed of a bony part and of a cartilaginous part. The bony part is made up of the two nasal bones. The nasal bones rest against each other medially to form the arch or bridge of the nose. Laterally, this arch or bridge of the nose is supported by the frontal processes of the maxillary bone. At the root of the nose the nasal bones are mortised into the nasal processes of the frontal bone. At this articulation the nasal bones are solid and heavy. Viewed from without, each nasal bone is first concave and then convex. At the junction of these two bends the nasal bone is thin, and this is the site where fracture most frequently occurs.

The cartilaginous framework is composed of the upper and lower lateral cartilages.

Upper Lateral Cartilages.—The upper lateral cartilages are not separate cartilages at all. Actually they are winglike lateral prolongations from the anterior edge of the septal cartilage. The medial edge of the upper lateral cartilage is continuous with the entire anterior edge

of the cartilaginous septum. This medial edge may extend caudad to the caudal anterior end of the septum, or it may occasionally end short of the caudal end of the septum. As long as the upper lateral cartilages are united at their medial margins, they support the nose throughout their entire length. Naturally, only if they end short of the septum or if they are separated from each other does the septum offer support to the areas in question. It is in such cases that one must be on one's guard against a saddle nose when a submucous resection is done. Each wing of the upper lateral cartilage is usually roughly triangular in shape. The base of the triangle is thus the anterior edge of the

UPPER LATERAL MAY END SHORT OF TIP OF SEPTUM
UPPER LATERAL SLIGHTLY BEYOND APERTURA PYRIFORMIS
UPPER LATERAL CAUDAL TO END OF SEPTUM

Fig. 1.—Relationships of upper lateral cartilages may vary.

septum. The cephalic part of the upper lateral cartilage is attached firmly by fibrous tissue to the medial aspect of the processus pyriformis. Occasionally the upper lateral cartilage may be quadrilateral. It is thickest at the septum and thins out laterally. The ectal surface is usually flat; occasionally it may be either concave or convex.

The cephalic edge of the upper lateral cartilage is always underneath the nasal bone medially and underneath the frontal process of the superior maxilla laterally and may extend in this relationship for a distance varying from 1 mm. to almost the entire length of the nasal bone. Because of this anatomic relationship, when the soft structures of the nose are elevated from the nasal framework one must seek the

caudal edge of the nasal bone and then proceed anterior to it. Otherwise, if one followed the anterior surface of the upper lateral cartilage one would slip underneath the nasal bone and then penetrate into the nasal cavity.

Invariably the caudal edge of the upper lateral cartilage first runs for a distance of about 5 to 10 mm., paralleling and almost hugging the septum in a direction slightly dorsal to the ventral edge of the septum. I believe that this is an important relationship to retain, inasmuch as it forms a nasal cord, just as in the larynx there is a vocal cord. The remainder of the caudal edge of the upper lateral cartilage then swings laterally and cephalad toward the apertura pyriformis.

The upper lateral cartilage is usually attached to the septum at an acute angle. It has a natural springiness or elasticity. When the pars compressor of the nasalis muscle contracts, the upper lateral cartilage is pressed against the septum. When the muscle relaxes, the upper lateral cartilage, of its own inherent elasticity, recoils away from the septum and the aforementioned angle is reestablished. The maintenance of this angle is important in quiet nasal breathing. Rhinoplasty, to be physiologically sound, must conserve this angle, and it must create this angle if it does not exist. This is accomplished by placing a loose petrolatum pack at the site of the angle when the rhinoplasty is completed.

In the nasal vestibule the anterior caudal part of the upper lateral cartilage crosses the dome of the vestibule like a shelf. This shelf helps slice the air stream into more surfaces to be physiologically acted on.

The upper lateral cartilage is surrounded by a sheath of perichondrium. Anteriorly, part of this sheath is reflected onto the posterior surface of the lower lateral cartilage, becoming continuous with the perichondrium of the latter. This sheath is an aponeurosis-like structure through which the initial incisions are made in rhinoplasty.

Lower Lateral Cartilages.—The lower lateral cartilages can best be visualized by imagining a teaspoon bent in the shape of a horseshoe with the handle somewhat pulled forward. The spoon-shaped part is the lateral crus. It gives the tip its shape and support. The handle is the medial crus. In the nose the two crura are connected by a dome-shaped expansion of the lower lateral cartilage. The variations in shape of the lower lateral cartilages are legion. The cephalad margin of the lateral crus rests on the upper lateral cartilage. The two medial crura and their surrounding structures form the columella of the nose.

Mobile Nasal Septum.—The columella is freely movable on the cartilaginous septum. This motility is made possible by the membranous septum.

The membranous septum consists of a tough elastic membrane connecting the free end of the septum and the columella. It is triangular in shape, with the base at the under surface of the lower lateral cartilage. The brevity of this description of the membrane should not belie its importance. It is one of the most important structures in rhinoplasty, since on its treatment depends the success or failure of many rhinoplasties.

Fixed Nasal Septum.—The rhinologist is familiar with the relationship of the cartilaginous septum to the perpendicular plate of the ethmoid

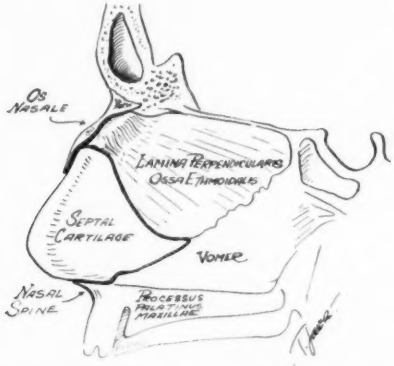

Fig. 2.—The septum. Note cartilaginous septum under nasal bones, irregularity of thickness of nasal bones and nasal spines and cartilaginous septum ventral to them.

and to the vomer. It is noteworthy that although the ventral aspect of the septum may be thin in the vicinity of the cribriform plate the septum may be post-traumatically as much as one-half inch (1.27 cm.) in thickness underneath the nasal bones. Attention should be called to the folding screen arrangement by which the caudal edge of the cartilaginous septum rests in the groove of the vomer. The ridge and angulations frequently seen at this site are thus easily visualized as the natural result of a crushing injury. Proceeding ventrally, after the caudal edge of the septal cartilage leaves the vomerine and maxillary grooves, it lies unsupported between the nasal spines for a distance of about 4 mm. The so-called free margin of the septum then proceeds in a graceful curve cephalad and ventrally toward its union with the upper lateral cartilages.

Nasal Spines.—The nasal spines are bony, pointed, prowlike projections of the anterior medial ends of the maxillary bones. They are strong. They point directly forward and at times make a prominance in the upper lip at the nasolabial angle. This is most noticeable when the patient smiles. Although the septum is frequently dislocated from its position between these spines by trauma, the spines themselves are rarely broken.

It is my opinion that the muscles of expression about the nose play an important part in nasal physiology. There seems to be some confusion in the various textbooks about these muscles and their actions. Dr. Raven, of the American Museum of Natural History, gave me permission to view and to study his most painstaking

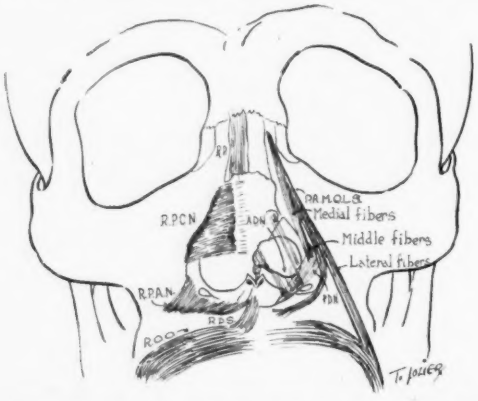

Fig. 3.—Muscles of expression about the nose. *R. P.* indicates right procerus muscle; *R. P. C. N.* right pars compressor of the nasalis muscle; *R. P. A. N.* right pars alaris of the nasalis muscle; *R. O. O.* right orbicularis oris muscle; *R. D. S.* right depressor septi of the orbicularis oris muscle; *A. D. N.* anterior dilator nares muscle; *P. D. N.* posterior dilator nares muscle, and *P. A. M. Q. L. S.* pars angularis of the quadratus labii superioris muscle.

and skilful dissection of the facial muscles of an adult gorilla. A careful study of the paper of G.H.S. Lightoller [5] titled "Facial Muscles" has yielded much information about the nasal muscles and nasal physiology. Due credit must be given to Lightoller for his research in the physiology of sound production, especially his interpretation of the action of the nasalis muscle in the production of the sounds "P," "B" and "M." His photographs of faces in action are well worth study,

5. Lightoller, G. H. S.: Facial Muscles: The Modiolus and Muscles Surrounding the Rima Oris with Some Remarks About Panniculus Adiposus, J. Anat. **60**:1-85, 1925-1926.

for it is his work which for the first time described the facial muscles as actually seen in action.

Procerus Muscle.—These muscle fibers arise from the bone at the nasofrontal articulation. They proceed cephalad and are inserted into the skin between the eyebrows. Their contraction causes wrinkles of the forehead between the eyebrows.

Nasalis Muscle.—The nasalis muscle is composed of two parts, the pars compressor and the pars alaris.

Pars Compressor: The pars compressor is a thin flat muscle. It has its origin from the maxillary bone lateral to the apertura pyriformis. It inserts into an aponeurosis on the ventral aspect of the nose. This aponeurosis is a common insertion for both pars compressors and thus helps make of the compressor muscles a nasal sphincter. There is a bursa underneath this aponeurosis. As the pars compressor runs ventromedially it crosses the cephalic part of the ala and then it crosses the upper lateral cartilage. Its contraction thus presses (1) the cephalic part of the ala inward and (2) the upper lateral cartilage toward the septum. A strong contraction of the pars compressor is necessary when saying "P" or "B." It is also strongly contracted in the crying infant. It is not so strongly contracted when "M" is voiced. This strong contraction of the pars compressor presses the part of the upper lateral cartilage just mentioned as the nasal stop against the septum. It helps create enough air pressure in the nose and mouth to burst the lips apart when such sounds as "P" "B" or "M" are voiced.

Pars Alaris: The pars alaris is a fan-shaped muscle. It arises from the maxillary bone at the dorsal and canine margin of the apertura pyriformis. Its lateral fibers insert into the caudal part of the ala. When these lateral fibers contract they pull the ala downward and outward. The pars alaris also sends fibers to the mobile part of the septum, being attached to the medial crura of the lower lateral cartilage, particularly to the winglike lateral flarings of the ends of the medial crura. Contraction of these fibers pulls the mobile septum downward. Here must be mentioned an upward extension of the muscle orbicularis oris called the depressor septi, which when it contracts also helps pull the mobile septum downward. Occasionally this depressor septi muscle is so prominent that it creates a webbing at the nasolabial angle. Its antagonist is the procerus muscle.

Other Muscle Groups.—To understand the movements of the alae three more muscle groups must be studied.

1. Pars angularis of the quadratus labii superioris muscle. This angularis muscle arises from the frontal bone and the frontal process of the maxillary bone. Its most medial fibers proceed downward and

are inserted into the cephalic part of the ala. When these medial fibers contract they pull the ala upward. They are the antagonists of the lateral fibers of the pars alaris of the nasalis muscle. The middle fibers of the angularis muscle proceed downward and outward and then curve around the ala and are inserted into the upper lip near the philtrum. When these medial fibers contract they help to bring the ala medially and thus narrow the nostril. The lateral fibers of the angularis muscle are inserted into the angle of the mouth. It can thus be understood that the dominant action of the angularis muscle, in smiling or laughing, is to pull the ala upward and sometimes to cause a crease of the upper lip. 2 and 3. Anterior and posterior dilator nares muscles. These are two muscles reaching to the rim of the nostril, influencing its size and shape. The anterior dilator nares arises from the ectal surface of the lateral aspect of the lower lateral and upper lateral cartilages and inserts into the rim of the nostril. The posterior dilator nares muscle arises from the nasal notch of the superior maxilla and from the lateral sesamoid cartilages and inserts into the skin and the outer edge of the rim of the nostril. By their action they tend to dilate and evert the nostrils.

The innervation of this entire muscle group is by the facial nerve. To quote from Lightoller:

> With the caput angulare pulling the ala nasi cranially and the pars alaris muscle nasalis pulling caudally and laterally the ala nasi, and caudally the inferior border of the nasal opening and septum mobile, we can see that the triangular exterior nares is very considerably modified in shape and made to approach the circular. This gives, of course, the greatest possible aperture.
>
> From the above it follows that to increase the capacity of the nasal airway there is primarily a widening of the anterior nares and secondarily a widening of the post vestibular area. To decrease the capacity of the nasal airway there is a primary narrowing of the post vestibular area and a secondary narrowing of the external nares.
>
> Depression of the nasal septum if it have any effect at all would rather be to widen the airway.

In forced or strained breathing the anterior and posterior dilator nares muscles are in a state of contraction so that, in addition to the aforementioned rounding, the nares are also slightly everted.

The pars compressor of the nasalis muscle, by controlling the space between the septum and the upper lateral cartilage, helps control the amount and the shape of the air current entering the nose proper.

It is at once apparent that since these muscles are so important in nasal physiology they must be visualized and their action understood when a rhinoplasty is being done. This can, in general, be best accomplished by always working close to the nasal framework.

As far as the innervation of the nose is concerned, there are three intranasal areas and three subcutaneous areas of importance in this

discussion. Anesthesia applied to these areas is sufficient for the operations here discussed. These areas are intranasally approximately at the anterior and posterior ends of the middle turbinate and at the nasopalatine foramen; subcutaneously the areas are at approximately the midpoints of the cephalad and caudad extremities of the nasal bone and at the infro-orbital foramen.

Fig. 4.—Type of deformity of nose and septum depends on direction and force of trauma.

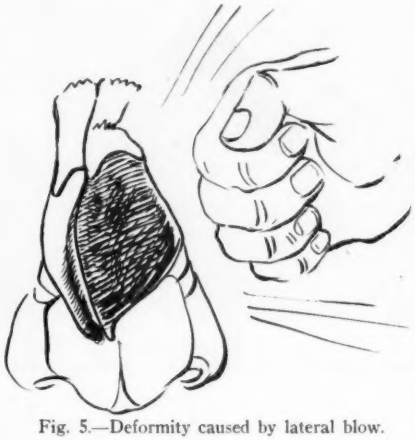

Fig. 5.—Deformity caused by lateral blow.

EFFECT OF TRAUMA

The effect of trauma on the framework of the nose in general and on some of the structures in detail will now be considered.

Figures 4, 5 and 6 show the result of injury to the nose (1) at the bridge, (2) at the upper lateral cartilages and (3) at the tip.

A deviated septum or a deformed nose, or both, can interfere with the function of the nose. There are instances in which a submucous resection alone or a rhinoplasty alone will improve nasal function. There are other cases in which both operations are necessary before satisfactory results can be achieved. The rhinologist is therefore confronted, in each case that requires operative intervention, with the necessity of first making a decision whether to perform a submucous resection alone, a rhinoplasty alone or both a submucous resection and a rhinoplasty. If both procedures are necessary, shall the operations be done separately or both at one operative session? If done

Fig. 6.—Dislocation of septum caused by trauma at the tip.

separately, which operation shall be done first? It is my opinion that whatever decision is made should be based on a thorough knowledge of the anatomic derangement and physiologic dysfunction found in each case. One should then be guided by surgical judgment and experience rather than by rule of thumb in how best to repair the damage found.

How does one proceed to look for anatomic and physiologic abnormalities? First, one inspects the exterior of the nose during quiet breathing. Varying degrees of alar collapse during inspiration yield the following information:

1. The columella may be too broad and obstruct the nostril.

2. The nostril may be too narrow and slitlike and thus cause abnormal variations of pressure within the nostril.

3. The structures of the roof of the vestibule may be atrophic or inadequately supported.

Then one examines the interior of the nose with and without mucosal shrinkage. This will yield much information familiar to the rhinologist.

When rhinoplasty is contemplated, a study of photographs of the face is important both for making a diagnosis of the type of nasal deformity and for achieving face balance. The set of photographs

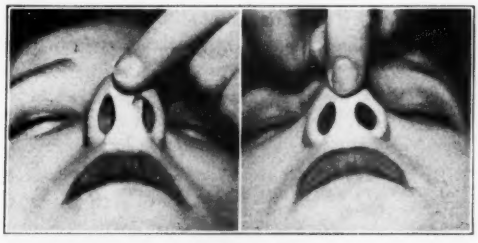

Fig. 7.—Nostril view—dislocated septum before and after correction.

Fig. 8.—Profile view—deformity of nose and dislocated septum before and after correction. Note correction of retracted columella by use of columellar cartilaginous strut.

employed is taken as follows: (1) one full view, (2) both profile views and (3) one nostril picture taken from beneath.

Aufricht [6] suggests an extra set of pictures taken during laughter, for the reason that many deformities are then exaggerated. The photographs are usually printed one-half face size, except for the nostril

6. Aufricht, G.: A Few Hints and Surgical Details in Rhinoplasty, Laryngoscope **53**:317-335 (May) 1943.

view, which is made natural size. The face balance here described is that taught by Dr. Samuel Foman.[7]

In the full face view the nose normally occupies the middle third of the distance from the hair line to the bottom of the chin. At the alae the outer margins should fall within perpendicular lines dropped from the inner canthi. The midpoint of the root of the nose, the midpoint of the tip of the nose and the midpoint of the philtrum should be in the same perpendicular line. This perpendicular line should bisect the nose.

The profile angle of the nose should be about 30 degrees. The angle between the columella and the lip or the nasolabial angle should be about 90 degrees or more. In profile view the columella should extend slightly beyond the outer rim of the nostril.

Fig. 9.—Same case as in fig. 8. Semiprofile view before and after correction. Note correction of hump and of cleft tip.

The nostril view yields information as to the width of the columella, the size and shape of the nostrils, the bulkiness of the tip and the flaring of the alae. A plan for the correction of any deformity and a pattern of tissues to be excised can be made from such a study.

A careful study of such photographs combined with a rhinoscopic examination helps one visualize what happened to the structures injured. With this knowledge one can better restore the structures to a normal appearance and therefore a normal function.

SEQUENCE OF OPERATION

Now, with knowledge obtained from the sources mentioned combined with a pertinent history, one can better decide on the sequence of operation when both a submucous resection and a rhinoplasty are indicated.

7. Foman, S.: Personal communication to the author.

1. A submucous resection should precede a rhinoplasty when a deviation of the septum exists with pathologic changes which may contraindicate a rhinoplasty, such as the existence of (*a*) purulent sinus infection which the septum does not permit to drain satisfactorily, (*b*) nasal polypi with chronic sinusitis or (*c*) thickening or deflections of the caudal half of the septum which may interfere with an adequate osteotomy.

2. The submucous resection should be done after a rhinoplasty (*a*) when extensive shortening of the nose is necessary—one thus assures tip support; (*b*) when extensive removal of the hump is necessary—occasionally a high cartilaginous deflection may be simultaneously removed, and (*c*) when the upper lateral cartilages end short of the caudal end of the cartilaginous septum. One thus eliminates the danger of a saddle nose following a too thorough submucous resection distal to the upper lateral cartilages and then not having upper lateral cartilage long enough to repair the defect.

3. A submucous resection must be done simultaneously with a rhinoplasty (*a*) when the septum is so thick at its ventral third that it interferes with adequate olfaction when the nose is narrowed; (*b*) when the septum makes infracture with adequate narrowing impossible; (*c*) when infracturing results in gaps created by a septum which is transverse to the midline and which cannot be forced into a midline position simply by thumb pressure, and (*d*) when a nasal deformity is accompanied with a dislocation of the septal cartilage which obstructs the nasal vestibule.

For the correction of the septal cartilage which obstructs the nasal vestibule the Kayser procedure advocates in part the following (from Kayser's article [1]): If a hump is present, it is removed and

> . . . the upper lateral cartilages are completely freed from their septal attachments. One usually finds that as soon as the fragment of cartilage is liberated from its marginal attachments it is drawn into an unnatural position beyond the mid-line because of the scar contracture of the mucoperichondrium attached to the other surface of this cartilage fragment. It is in this instance that it is necessary to excise the deflected cartilage entirely. . . . The mucoperichondrial flaps are undermined both from the remaining septum and from the floor of the nose until enough slack is created to permit the flaps to stay in the mid-line when complete healing has occurred. . . . The upper lateral cartilages . . . are then sewn together. . . .

The operator knows that these upper lateral cartilages are themselves supported from the ental surface of the nasal bones. He must always insure adequate support of these nasal bones. Otherwise when a hump is removed and too much perpendicular plate of the ethmoid is taken away, the entire outer framework of the nose may collapse as soon as an osteotomy is completed. In some instances the upper

lateral cartilages themselves may be too inelastic or of poor consistency, so that in the judgment of the operator they may not be satisfactory for the support of the ventral aspect of the nose. In such instances the operator must improvise other forms of support.

Mention was made of the nasal musculature. When one understands the repeated pulls on the mobile septum and the alae caused by these muscles it becomes apparent how futile some sutures, such as the orthopedic suture, ultimately become.

CONCLUSION

Detailed knowledge of anatomy, experience and improvisation are the keystones for progress in surgery of the septum.

Abstracts from Current Literature

Ear

EVALUATING THE EFFICIENCY OF GROUP SCREENING TESTS OF HEARING. HAYES A. NEWBY, J. Speech & Hearing Disorders **13**:236 (Sept.) 1948.

The author describes a simple technic which can be applied in study of the efficiency of a group screening test. In examining the efficiency of a group pure tone screening test, the author tested every fourth child individually. The more subjects were tested individually, the better was the measure of efficiency obtained. No fewer than 50 subjects or 100 ears should be tested individually in any group. Newby found it useful to combine children of the eighth and seventh grades; those of the sixth, fifth and fourth grades, and those of third and second grades in a sampling test of group screening in the school system. A convenient method of examining graphically the data thus obtained is the use of a tetrachoric table. This table is used not to obtain a coefficient of correlation, but merely to portray the data in a meaningful fashion. Once a criterion of a significant hearing loss has been properly established, it is easy to adjust the efficiency of any group test by means of a tetrachoric table. If no amount of juggling of the criterion of significance of the group test results in reducing the percentage in any particular cell used to indicate success without raising the percentage in another cell used to indicate failure, then the group test is not operating with satisfactory efficiency.

PALMER, Wichita, Kan.

MEDICAL TREATMENT OF MÉNIÈRE'S SYNDROME. JOHN J. RAINEY, New York J. Med. **48**:1833 (Aug. 15) 1948.

Rainey reports excellent results in 83 per cent of 121 patients seen at Troy Hospital since 1940 who were treated with intravenous injections of histamine phosphate.

Attacks of Ménière's syndrome are said to be brought on by release of histamine from body sources. The syndrome is characterized by dizziness with a turning sensation, deafness in one or both ears, nausea or vomiting or both. It is a disorder of the vestibular labyrinth, for which the phrase "labyrinth hydrops" has been suggested. Rainey is opposed to use of that term. He states that the earlier the diagnosis is made and treatment is instituted, the more likely is recovery to be complete. In fact, "most patients get well with the passing of time." He reports 2 cases as representative of the group. About 1 patient in 7 requires intravenous administration of histamine. Three patients had resections of the auditory nerve, but only "improved" (in the presence of total deafness). One had had a nerve resection twelve years before, with return of symptoms five and one-half and twelve years later.

A salt-free regimen with ammonium chloride was of "no benefit." The newer drugs, diphenhydramine (benadryl®) and tripelennamine (pyribenzamine®) should be used cautiously, as they may "confuse the issue."

Administration of histamine phosphate into a vein is time consuming, requiring from one and one-half to two hours. However, if the physician's diagnosis is correct, he will have in histamine phosphate used intravenously the best treatment at present available.

Abstracter's Comment.—Some readers may criticize this laudatory report because of the apparent inconsistency between the statement that most patients

get well with any or no treatment, and that about 83 per cent get well with histamine technic. However, a desperate patient will demand that "something be done."

VOORHEES, New York.

Larynx

CONGENITAL MALFORMATION OF THE ESOPHAGUS WITHOUT TRACHEO-ESOPHAGEAL FISTULA. JAMES R. LISA AND HELEN M. TAYLOR, New York State J. Med. **48**:1622 (July 15) 1948.

A boy, born Jan. 25 1946, died in the fiftieth hour. The first feeding was given six hours after birth and was repeated, but with each there were cyanosis, gasping and regurgitation. Diagnosis at autopsy showed atresia of the middle third of the esophagus without tracheobronchial fistula. Acute bronchopneumonia was also present. A review of the literature shows that this condition occurs in 1 of 20,000 births (San Giovanni Maternity Hospital, Rome). At Bellevue Hospital (New York) only 1 case was found in sixteen years. At the City Hospital, Welfare Island, 3 cases were noted in 5,158 autopsies. The total reported number in the literature is about 400. "Total absence of the esophagus . . . has not been reported in the last twenty-seven years . . . There have been many cases of esophageal atresia without fistula." Heredity plays no part, but hydramnios should call attention to a possible congenital anomaly. Roentgenography is of value in outlining the upper pouch, taken with barium sulfate or iodized oil U. S. P. Furman recommended esophagoscopy without these agents. Surgery offers a chance, but attempts years ago were unsuccessful. Success depends on early diagnosis, prompt operation and avoidance of any complication, such as pneumonia. Stenoses may be dilated in older infants and children. The authors offer fifty-eight references from the literature.

VOORHEES, New York.

Nose

SOME PITFALLS IN SINUS SURGERY. J. JEROME LITTELL, Laryngoscope **58**:396 (May) 1948.

Otolaryngologists stand "before the bar of public opinion for [their] too frequent failure, in the past, to satisfy the patient that he has had a satisfactory degree of lasting improvement from surgery . . ." Littell gives rules to be followed and factors to be considered in order to achieve satisfactory surgical results. Many of the sinal operations are discussed in the light of the mistakes that should be avoided and the results that should be attained.

HITSCHLER, Philadelphia.

BENIGN CYSTS OF THE ANTRUM ORIGINATING FROM THE JAW OR TEETH. W. A. NEWLANDS, Laryngoscope **58**:402 (May) 1948.

Newlands presents a comprehensive report on benign cysts of the maxillary sinus originating from the jaw or teeth. Their origin, classification, diagnosis (including roentgenologic), treatment and many other aspects are discussed in some detail. Two cases, one of a dentigerous and one of a radicular cyst, are presented; the difference between these two types is stressed.

Hitschler, Philadelphia.

SURGICAL TREATMENT OF ETHMOIDITIS. JAIME SALA LOPEZ, An. de oto-rino-laring. d. Uruguay **17**:77, 1947.

Sala Lopez discusses the anatomic problems involved in the treatment of acute and chronic ethmoiditis and presents a chronologic discussion of various operative procedures that have been advocated for control of these conditions. He states that one should remove the cause of insufficient drainage from the middle meatus. Whatever procedure is indicated should be resorted to, whether it be removal of the anterior portion of the middle turbinate body or a submucous resection. These procedures should never be undertaken in the presence of an acute infectious process. If the ethmoid sinuses have become a septic cavity and have not been drained properly, they may act as a focus of infection. Proper drainage of these sinuses is always more advantageous then curettage, for after all, even though the ethmoid sinuses may be perfectly curetted, if drainage and aeration are insufficient they can always be a source of reinfection. The author believes that the transantral approach to the ethmoid sinuses assures an exposure for the most complete drainage, particularly when the posterior ethmoid and sphenoid sinuses are involved. This is also valuable in the cases of fronto-ethmoid sinusitis. He is of the opinion that an external approach should be used only if a transantral operation has not afforded sufficient drainage and aeration. Attention to the maxillary sinuses is of extreme importance, and treatment of these sinuses should be included with that of other sinuses involved.

PERSKY, Philadelphia.

Miscellaneous

RELATION OF DOSAGE TO STREPTOMYCIN TOXICITY. RALPH TOMPSETT, Ann. Otol., Rhin. & Laryng. **57**:181 (March) 1948.

Streptomycin is a valuable agent in treating many acute and chronic infections for which no other drugs are available; but frequent and severe toxic reactions occur from its use, and it may produce rapid emergence of resistant strains of organisms. The diseases for which the drug is effective are, in general, serious infections with a high rate of mortality and for which no other effective antibiotic agent is available. Because of this and the knowledge that the organisms rapidly develop resistance to the drug, there has been a tendency to administer large doses of it. Also, it has been thought that, since the drug was so rapidly absorbed and eliminated and that maintenance of constant levels was necessary, frequent injections must be given. The author presents evidence that neither the frequent injections nor the maintaining of constant levels is necessary. His observations would suggest that the rational regulation of dosage schedules should be based on the body weight of the patient and that, probably, long intervals may be allowed between injections. The author discusses some of the various complications caused by the drug, including allergic manifestations. In 5 cases in which the latter appeared, therapy was able to be continued by the use of another drug, dihydrostreptomycin, which is closely related to streptomycin and which, so far as tested to the present time, appears to have similar antimicrobic activity. Organisms sensitive to streptomycin are also sensitive to this drug, and those resistant to one are resistant to the other.

M. V. MILLER, Philadelphia.

REHABILITATION OF THE AURALLY HANDICAPPED THROUGH THE STUDY OF SPEECH READING IN LIFE SITUATIONS. BORIS V. MORKOVIN, J. Speech Disorders **12**:363 (Dec.) 1947.

In training for speech reading, stress should be placed on the complete stimulus pattern rather than on the movements of the lips or other single items. Speech

reading is most successful when it is taught on the basis of life problems and the necessity for solving them. All the senses are interlocked in their modalities, and successful teaching is done by auditory-visual-kinesthetic cooperation. Motion picture films are helpful in teaching speech reading. Two illustrative cases are presented.

PALMER, Wichita, Kan.

A PROGRAM FOR THE SPEECH-INHIBITED CHILD. AMY BISHOP CHAPIN and MARGARET CORCORAN, J. Speech Disorders **12**:363 (Dec.) 1947.

The term delayed speech has been used too often to include all kinds of immature speech development. Subjects accepted for this study were screened by excluding the mentally deficient, autistic, prepsychotic children, children with cretinism and mongolism and children with deterioration and gross damage to the brain. The children selected, therefore, showed evidence of normal, intelligent behavior and passed all neuropsychiatric examinations with a diagnosis of some form of emotional-environmental retardation or suspected aphasia. Sixteen children were in the study, with an average age of 5.2 years. That the children were in a nursery school made it possible to overcome their fears and feelings of inadequacy and to develop social sufficiency. Speech was stimulated very carefully. Each child was addressed as though he were expected to talk. A great deal of work was done with the parents, explaining by lecture and individual conferences the problems involved. Of the 16 children, 12 were either dismissed as having attained normal speech for their age level or were accepted at regular schools. Two needs were discovered in this study: first, an adequate pamphlet for parents and, second, reliable diagnostic procedures. The author states that 45 per cent of children with so-called delayed speech referred to the clinic in a six month period had no endocrine disturbances and were not deaf, feebleminded, autistic or prepsychotic, but were merely speech-inhibited normal children.

PALMER, Wichita, Kan.

VISUAL AIDS TO SPEECH IMPROVEMENT. WILBERT PRONOVOST, J. Speech Disorders **12**:387 (Dec.) 1947.

The author discusses the use of the oscilloscope for showing differences in rate, duration and pitch in speech. The oscilloscope is also used in some work with defective voices when too large a crescendo occurs, as in the "glottal attack." The voltmeter, decibel indicator and similar devices are useful in regulating volume. An apparatus which will measure nasality directly by activating an amplifier is mentioned, and a speech game for children, in which letters are mounted behind a bulb system with nine windows, is discussed.

PALMER, Wichita, Kan.

THE WISCONSIN CLEFT PALATE PROGRAM. GRETCHEN MUELLER PHAIR, J. Speech Disorders **12**:410 (Dec.) 1947.

The author reviews briefly the history of the incidence of cleft palate. In the state of Wisconsin, for a period of ten years, between Jan. 1, 1935 and December 31, 1944, 1 case of cleft palate occurred in every 770 births. The percentage of births with cleft palate was greater for the nonglaciated areas than for the glaciated. A larger number of children with cleft palate were born during the summer season (July, August and September), than during the other months. The incidence of cleft palate increased slightly during the war years.

Mothers in the age group of 20 to 24 years produced many more children without cleft palate, while the groups of 35 to 39, 40 to 44 and 45 plus produced

proportionately more children with cleft palate. Cleft palate occurred more frequently in male children, there being 61.5 per cent of boys in the group. In the state of Wisconsin, out of 226 children with cleft palate, 9.73 per cent were twins, while only 2.15 per cent of the normal total live births were twins. A slightly higher incidence of cleft palate occurred in rural areas, and a higher percentage of cleft palate occurred in premature children. There is a slight tendency for the condition to occur more frequently on the left side.

Since 1943, a program of surgical correction, use of prosthetic appliances and orthodontic treatment together with correction of speech pathology, has been conducted in the Wisconsin Orthopedic Hospital. The Bureau of Handicapped Children in the State of Wisconsin has a medical social worker at the Wisconsin Orthopedic Hospital who handles the social problems which arise. A child welfare consultant works during the summer, finding boarding homes and supervising such social problems.

PALMER, Wichita, Kan.

THE VETERANS ADMINISTRATION PROGRAM IN THE FIELDS OF AUDIOLOGY AND SPEECH CORRECTION. MERLE ANSBERRY, J. Speech & Hearing Dis. **13**:115 (June) 1948.

Approximately 65,322 veterans of World War II with service-connected hearing losses or potential speech disabilities are now on the rolls of the Veterans Administration. Of all veterans drawing compensation for disabilities resulting from service in the recent war, 2.5 per cent have been adjudicated for hearing disabilities; 1.2 per cent of the same group have disabilities which make them potential candidates for speech rehabilitation. The Veterans Administration provides rehabilitation services through the Physical Medicine Rehabilitation Division and the Neuropsychiatry Division. Thirty contracts are at present in effect with professionally qualified speech clinics. The mental hygiene clinics of the Veterans Administration are also providing speech therapy by qualified persons. A training program is under way at Ohio State University and will be extended to other universities, probably in the near future, for qualified personnel in the field.

PALMER, Wichita, Kan.

ALLERGY IN OTOLARYNGOLOGY. FRENCH K. HANSEL, Laryngoscope **58**:652 (July) 1948.

A need exists for a method of treating hay fever that does not involve a long, and sometimes dangerous, series of injections. Each case must be studied individually. Diagnosis must include a careful history and examination, including the nasal secretions for the presence of eosinophils. Complete skin testing is necessary, and details are given explaining the procedure and the reactions to be expected. The coseasonal method of treatment (that of giving injections every one to three days during the hay fever season) is preferred by Hansel. Fewer injections are needed, and therefore no dangerous reactions are apt to occur. Oral administration of pollen extract instead of hypodermic injection is now being tried, with some success. No definite conclusions as to the exact efficacy of this treatment can as yet be drawn.

HITSCHLER, Philadelphia.

Society Transactions

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

John R. Lindsay, President

Lawrence J. Lawson, Secretary

Regular Monthly Meeting April 5, 1948

The Development of the Auditory Ossicles and the Temporal Bone: A Summary of Research. Presented by BARRY J. ANSON, PH.D., of the department of anatomy of Northwestern University Medical School.

Various phases of our otologic investigation, pursued divisionally, have already been described in a series of journal articles. Now we are able to correlate the once separate parts of the study and, as a consequence, to report on crucial steps of the development of the ossicles, of the otic capsule and of the extracapsular portions of the temporal bone.

Our observations are based on a study of approximately 300 series, chiefly in the collections at the University of Wisconsin and at the Northwestern University Medical School.

In general it may be said that maturity of gross form and size and of histologic structure is attained by the ossicles and the otic capsule far earlier than was once supposed. In fact, their development is so rapid that adult dimensions and configuration of the ossicles and the capsule are attained when one might expect them to be fetal. Equally remarkable is the fact that morphogenesis takes place in such a way as to violate some of the important conventions of the ossification process.

At 25 mm. (or 8 weeks) the cartilaginous stapes is still a ring. At 50 mm. (11 weeks) the features of a stirrup are acquired. At 126 mm. (17 weeks) the base is marginally lipped, the head foveate so that it may articulate with the incus, the posterior crus longer and bowed—features which foreshadow adult configuration even before ossification has begun.

In the 150 mm. fetus (20 weeks) the single center of ossification has appeared on the obturator surface of the base. At 160 mm. bone has spread across the base, but not across the neck of the stapes. Concurrently, newly formed bone is being removed on the inner, or obturator, surface, but remains intact on the outer aspect. Erosion is deep, into the calcified cartilage. Bone continues its advance, forming a complete ring by the 180 mm. stage (23 weeks). Since no epiphyses occur, no further growth takes place; adult size has been attained. However, removal of bone continues. Within another month and a half, that is, in the 245 mm. fetus (29 weeks), mature form is virtually acquired.

It is essential to keep in mind the steps in the morphogenesis of a typical long bone. A human tibia will serve as an example. The cartilage "model" of a tibia suggests in a general way the form of the future bone. A shaft, or diaphysis, ends in slightly enlarged extremities which are the future epiphysial areas. Ossification begins in the diaphysis, to produce an elongate collar of bone. Externally, through activity of the osteoblasts in the periosteum, the enveloping shell of periosteal bone lengthens and thickens. Internally, spicules of endochondral bone and associated marrow tissue replace the original cartilage. At this stage bone formation is

augmented by an auxiliary histologic mechanism for longitudinal growth, namely, the secondary ossification center. One center appears at birth, the other soon thereafter. In both, cartilage persists as a growing plate—distally until the person is 17 years of age, proximally through the nineteeth to the twenty-third year. When cartilage is replaced by bone, growth ceases. Cartilage remains only on the articular surface of each extremity. Thus it is that a tibia grows steadily until the person has reached manhood. In an adult person the tibia is thirty-six times as long as it was when it made its first appearance in cartilage.

A stapes differs strikingly in structure from a long bone: Its ossification takes place from a single center; once formed in bone, it does not become thicker—there being no external application of bone to the periosteal layer, negligible production of endochondral bone internally; the stapes does not lengthen after periosteal bone has become continuous around the obturator foramen (in the fifth month), there being no epiphysial, or secondary, centers for growth; unlike a typical long bone, one wall—that facing the obturator foramen—is removed continuously in the capital, crural and basal portions of the stapes; marrow in what would be the shaft, or diaphysis, of a long bone is completely removed and is replaced by mucous membrane and submucosal tissue. Thus, while a tibia continues to grow into the period of early manhood, the stapes attains adult dimensions before the fetus has reached the halfway mark in its intrauterine existence. Within another two month period thereafter the stapes attains adult form. Whereas a tibia becomes progressively heavier while it is maturing, into the twentieth year, a stapes loses bulk while attaining maximum dimensions and adult form in the fetus. A stapes is far less robust as a mature ossicle than it was as fetal cartilage.

The bony otic capsule differs from other bones in respect to form, structure and function. It houses the sensory organs of hearing and equilibrium. It is capsular, that is, a box, being, in the adult, a single bone (therefore unlike the cranium which is made up of many elements separable at the sutures). Unlike cartilage bones, it has ossification occurring at various sites—a total of fourteen. These centers ultimately fuse so completely that the lines of original junction are virtually lost.

The otic capsule is composed of three layers of bone, namely, the periosteal (outer), the endochondral (middle layer) and the endosteal (internal layer, surrounding labyrinthine spaces).

The middle layer of the capsule differs from that of a typical long bone in containing numerous small remnants of modified cartilage which remain prominent through life; these islands of modified cartilage, later surrounded by endochondral bone, are the so-called intrachondrial bone. Cartilage persists also as a partial lining of the fissular tract anterior to the vestibular window (fissula ante fenestram) and for the inconstant tract of shorter course situated posterior to the window (fossula post fenestram).

In a tibia much of the endochondral bone gives way to marrow; in the petrous part of the temporal bone, on the contrary, little or no marrow is found. In the petrosa portions of the fetal cartilage are converted into intrachondrial bone and are retained in this form.

The development of endochondral bone, of the periosteal and endosteal layers, and of their predecessor, cartilage, will now be described as encountered in a series of five selected stages in the morphogenesis of the otic capsule.

In the 126 mm. fetus (16 weeks) the capsule is still entirely cartilaginous. In the 183 mm. fetus (21 weeks) cartilage is present at the posterior portion of the canalicular capsule and around the fissula. Endosteal bone forms a thin shell for the labyrinthine spaces (cochlea, vestibule and semicircular canals); this layer

undergoes no further growth, since no epiphysial areas exist. Periosteal bone is peripheral, on the cranial and tympanic walls. Intrachondrial bone occurs between these, representing the middle layer; it possesses a spongious appearance, as there is more space than bone.

At 270 mm. (30 weeks) periosteal bone is a thickening stratum. It spreads to invade the canalicular part of the capsule. Endochondral bone now makes its first appearance—in the region of the superior semicircular canal. It begins to cover in the intrachondrial bone of the primordial osseous capsule. Destruction of the recently formed periosteal bone has begun (in the canalicular region); the mucous membrane of the antrum is already replacing osseous tissue and associated marrow.

In the term fetus the periosteal layer is thick in the cochlear region. Intrachondrial bone in the same region is reduced in relative amount as "replacement" (endochondral) bone is deposited on the intrachondrial spicules. Endosteal bone is unchanged. Through the formation of the endochondral bone marrow space has been reduced, relatively, forecasting ultimate fusion of the two osseous strata.

In an adult of 65 years periosteal and endochondral, or "replacement," bone together make up almost the total bulk of the capsule and of the bone deposited theron. The replacement bone is a combination of the intrachondrial and endochondral types; the former (intrachondrial) formed the "skeleton" on which the latter (endochondral) has been deposited. In the adult the intrachondrial bone persists, but the surrounding endochondral bone, now more abundant, has virtually obliterated the marrow spaces. This middle layer can be distinguished from the outer stratum.

All these changes take place without increase in dimensions of the capsule, after the five month stage. The endochondral bone is not destroyed internally, to be reformed progressively; it remains virtually unaltered. However, periosteal bone does thicken; deposited on the capsule, it embeds the latter in extracapsular osseous tissue—to increase the dimensions of the so-called petrous part of the temporal bone, and to bring to the latter the form it here presents.

The periosteal bone of the late fetal and the infantile ear does not long remain intact. In the fetus of 260 mm. (29 weeks) the advancing epithelium has reached the mouth of the developing antrum. Invasion is rapid, spread taking place from the tympanic cavity and the auditory tube, to surround (in some specimens) the carotid canal, to reach the apex of the temporal bone and to occupy the former site of bone between semicircular canals—thus displacing not only periosteal bone of extracapsular position but also some of the endochondral bone of the capsule itself, the original capsular tissue tending to remain intact. Pneumatization, in some specimens, is complete at the age of 3 years or earlier.

In the otic capsule the first of a total of fourteen ossification centers appears early in the fourth lunar month (120 mm.). Soon three are present. Others appear in rapid succession, enveloping the labyrinth, replacing cartilage. They have fused to form a complete box, or capsule, prior to the middle of intrauterine life (183 mm.). Thus, adult dimensions are attained at the same time that the stapes attains full size. In a short period of five weeks the osseous capsule has been formed. Growth is prevented by fusion of the numerous centers of ossification (without intervening zones of epiphysial nature) and by concomitant formation of endosteal shells for the canals and cochlea.

Thus, although the otic capsule of the midterm fetus is as large as it will ever be, it is neither histologically mature nor is it yet embedded in the extracapsular osseous tissue whose outline lends the typical form to the so-called petrous part of the temporal bone. In the core of the capsule, between periosteal and endosteal layers where marrow might be expected to occur, bone of endochondral character

is deposited on remnants of persistent, changed, cartilage. Once formed, such tissue is not removed as it would be in a typical long bone, to make way for marrow. When removal occurs, it is to permit mucous membrane to spread from the tympanic cavity or the auditory tube into part of the capsule. Concurrently, periosteal bone—added to the capsule in the latter half of fetal life—is similarly invaded to form the cells of the apical and related portions of the temporal bone.

DISCUSSION

DR. ELMER W. HAGENS: Dr. Anson has given the members of this society another of his illuminating papers on developmental anatomy of the ear. As otologists the members are fortunate in having in their midst someone who is not only capable but sufficiently interested to give them the results of his research in their field.

Some years ago I had the opportunity of studying the development of the human ear in a group of fetuses at the University of Chicago. My series was not nearly so complete as Dr. Anson's, nor did I have the opportunity to study the material in detail. However, I did find many of the structures he has mentioned, and can appreciate the labor and the care that has gone into his investigation. A number of the facts brought out may have a relation to clinical findings. For example, in the early stages of the development of the stapes the head is connected with the hyostapedial tissue from which the stapedius muscle and tendon are derived. In 1 of 4 cases of malformation which I have studied, the malleus, the tensor tympani muscle and the incus were present while the stapes and the stapedius muscle were absent. In a second case the malleus, the tensor tympani muscle and the incus were absent, while the stapes and the stapedius muscle were present. These findings can be correlated with the development of the ossicles as presented by Dr. Anson. Cases have been reported of malformation of the stapes, such that only one incus extending from the center of the footplate was found. I wonder if Dr. Anson has any idea as to how this could occur. I believe his finding of the double origin of the stapes is an important contribution; it makes the development of this ossicle much more understandable.

In development of the ossicles it is noted that the malleus and the incus contain marrow, while the stapes when fully developed does not. In examining sections of the temporal bone having a history of otitis media, I have found infection extending into the marrow cavity of the incus from the tunica propia of the mucosa. In other sections the stapes is found completely embedded in infected tissue, but the outlines of the ossicle can still be seen. Owing to the difference in anatomy, infection may affect the malleus and the incus in one manner and the stapes in another. Otosclerosis frequently involves the stapes, whereas, except for one or two reports in the literature, otosclerosis has not been found in the malleus and the incus. Whether the anatomy of the particular ossicle has anything to do with this is problematic, but one may speculate on it.

Study of the three layers of the bony labyrinth has assumed increasing importance because of the fenestration operation. Experiments are going on at present as to the best method of making the fenestra remain open, but the final solution is yet to come. Careful studies such as these of Dr. Anson's may reveal clues that will help in finding the answer to the problem of the closure of the fenestra.

It is interesting to note that while the petrosa is different from all other bones in that its enchondral layer contains cartilaginous rests and other islands of cartilage, yet this is the layer where otosclerosis usually begins. Furthermore, otosclerosis does not occur in other bones, nor has it been found in animals. Dr.

Bast, one of Dr. Anson's co-workers, has found the fissula ante fenestram only in the human temporal bone; also, he has noted that otosclerosis, starting at the "site of predilection," begins in the secondary cartilage formation about the fissula. Finally, the fissula becomes obliterated and, he feels, this may possibly produce deafness by obstructing the perilymph-filtering function of the fissula. Thus one finds otosclerosis—which is found only in the human patient and only in the temporal bone—beginning usually in tissue about the fissula ante fenestram, which is, itself, found only in the human being.

Whatever the true relationship and importance of these findings may be, it is evident that studies of the temporal bone such as these are of great interest to otologists, and may well lead the way to a solution of some of their baffling clinical problems.

DR. BARRY J. ANSON: In addition to thanking Dr. Hagens for his generous comments, I should like to emphasize one point. From the developmental standpoint the ossicles should not be regarded as bones which follow similar patterns of morphogenesis. The malleus and the incus resemble a miniature tibia; their developmental schema matches that of a typical long bone, despite the fact that they are derived from the branchial arch skeleton. The stapes, although likewise originating at least in part from an arch, does not conform to the "standard" pattern. The stapes (closely related in its basal portion to the otic capsule) undergoes profound alteration in form after it has been fully developed in cartilage; the bone of the ossicle is removed continuously on the obturator aspect with, concurrently, negligible production of intrachondrial bone within its crura. It loses approximately one half of its bulk while attaining adult form and size in the fetus. Its unusual developmental history would be expected to make a difference in any pathologic change which it might later manifest.

Intranasal Medication with Vasoconstrictors. Presented by DRS. O. E. VAN ALYEA AND ALLEN DONNELLY, of the department of otology, rhinology and laryngology of the University of Illinois College of Medicine.

An investigation was carried out at the Research and Educational Hospitals, of the University of Illinois, to determine the effects of nasal vasoconstrictors in cases of hypertensive, cardiac and thyroid disorders and diabetes mellitus. This investigation was prompted by the fact that the Federal Food, Drug and Cosmetic Act requires a "warning" on bottles containing ephedrine or vasoconstrictors of similar character. This warning reads: "Caution: Frequent or continued use may cause nervousness, restlessness or sleeplessness. Individuals suffering from high blood pressure, heart disease, diabetes or thyroid trouble, should not use this preparation except on competent advice."

This study was made on 11 patients with hypertension, 16 with cardiac disorders, 9 with thyroid disorders and 14 with diabetes. After a complete examination, the patient was placed in the head-low position on an examining table, and 5 drops of a vasoconstrictor was instilled into each nostril. The position was maintained for several minutes; then the patient was returned to the sitting position. Pulse and blood pressure readings were made at intervals up to forty-five minutes. The testing solution applied was one in common use, "neo-synphrine hydrochloride" (phenylephrine hydrochloride N.N.R.) in the strengths of 1 per cent and 0.25 per cent.

Results.—No untoward effects were noted, and it was thought by the investigators that absorption from the nasal membranes is entirely too retarded to be of consequence in the development of side effects, and that when such do occur it

could be assumed they were due to drugs that had been inadvertently swallowed and absorbed from the stomach.

In view of this probability and the results of the present investigation it is felt by the writers that in the intranasal application of vasoconstricting solutions no distinction need be made between patients who have and those who do not have organic disorders and that the warning called for by the Federal Food, Drug and Cosmetic Act is unwarranted. A substitute warning, should one be required, could well read: "For nasal medication only. Do not swallow."

DISCUSSION

Dr. Carl C. Pfeiffer: This courageous study by Dr. Van Alyea and his associates is meritorious and should be called to the attention of the Food and Drug Administration. They are a relatively small scientific group in Washington; they do not have on their staff any member of a specialty group such as this; they cannot make experiments. They may consult experts, but sometimes these may fall within the definition of an expert consultant for the Army; namely, "any S.O.B. from out of town." Nevertheless, one must point to the good things they have done. They have been instrumental in stopping the marketing of vasoconstrictors suspended in liquid petrolatum. They have prevented the toxic effects of many other drugs which would have been foisted on the unsuspecting public. They are not concerned with the therapeutic merit of a drug, only with its possible toxic effect.

With regard to nasal vasoconstrictors, the history of the use of these compounds has been rife with minor toxicity. Physicians know that epinephrine produces a reactive hyperemia which is worse than the primary condition. They know that "argyrol" (mild silver protein U. S. P.) may produce argyria and that it will also destroy the cilia of the nasal mucous membrane. They know that the use of ephedrine in oil may give rise to lipoid pneumonia in children when the oil drains into the lung. They know that nasal vasoconstrictors, particularly eucalyptus oil or menthol, will congest the nasal mucous membrane. "Chloretone" (chlorobutanol U.S.P.) paralyzes the nasal cilia. So there are still harmful medications on the market. The use of "privine hydrochloride" (maphazoline hydrochloride N.N.R.), as Dr. Van Alyea said, may produce reactive vasodilatation. Amyl nitrite or organic nitrites when applied to mucous membranes are promptly absorbed, but with the water-soluble vasoconstrictors there is decreased blood supply, and little is absorbed in the mucous membrane. The greatest systemic danger comes from swallowing vasoconstrictor compounds which are not destroyed in the stomach; phenylephrine is, ephedrine is not. Physicians have looked for vasoconstrictors which are without central stimulation.

Dr. Van Alyea's study has gone a long way in reassuring otologists that vasoconstrictors can be applied to the nasal mucus membrane without fear of complications as long as they are not swallowed. If this study is brought to the attention of the Food and Drug Administration, they will undoubtedly modify their labeling requirements.

Dr. Alfred Lewy: In view of the fact that one proprietary product was brought out prominently in this investigation and, further, in view of the fact that Dr. Pfeiffer has mentioned two others as having similar effects, I should like to ask if he has a more complete list of proprietary drugs that can be used without danger of their being absorbed from the stomach or of central stimulation. If one goes into the matter of proprietary medication at all, I do not think one should advance any product above another.

DR. JOHN R. LINDSAY: Dr. Van Alyea has mentioned that many patients have suffered from the prolonged use of "privine." It would seem reasonable that the authorities should warn against using such a drug for prolonged periods except under the advice of a physician.

DR. CARL C. PFEIFFER: Most of the proprietary products that are not specialties of given pharmaceutical houses contain an ephedrine salt, 1 to 3 per cent; 5 drops of a 3 per cent solution would provide about 10 mg. of the ephedrine salt, and, if applied often, would be an effective systemic therapeutic dose. Except for epinephrine, the only nasal constrictor which is now known to be destroyed to any extent in the stomach is "neo-synephrine hydrochloride" (phenylephrine hydrochloride N.N.R.). It is true that Frederick Stearns & Company Division had an elixir of this drug which they marketed, but this has been ineffective. Even their neo-synephrine hydrochloride solution supplied in ampules for intramuscular injection will be destroyed within a short time if any metal comes in contact with it. This occurs the first time a hypodermic needle is used to remove a dose from a vial. So vials of this solution should not be used for more than one or two weeks. That is true also of the metal atomizer; the active substance will be slowly destroyed unless an all glass atomizer is used.

DR. O. E. VAN ALYEA: As I mentioned in the paper, this is not a complete study. We hope to carry out a similar investigation using ephedrine. I doubt that we shall use "privine," but I think we should use ephedrine. The paper was not intended to impress the Federal Food, Drug and Cosmetic group, but we thought it a subject which would be good for investigative purposes in view of our experience with drugs applied locally in the nose.

Hand-Schüller-Christian Disease and Eosinophilic Granuloma of the Skull.

Presented by DR. HAROLD F. SCHUKNECHT, of the department of otolaryngology of the University of Chicago.

Eosinophilic granuloma, Letterer-Siwe disease, and Hand-Schüller-Christian Disease are now considered to be closely related conditions. They are thought to be clinical gradations of expression of a common basic disorder. There are two theories as to their cause; one holds that there is a disturbance of lipid metabolism, whereas the other regards the disturbance as an infectious process.

Letterer-Siwe disease is a rare, rapdily fatal form occurring before the age of 2 and is of little clinical significance to the otolaryngologist. Hand-Schüller-Christian disease is a less severe form of the disorder, having a 30 per cent mortality. The destructive skeletal lesions which characterize this disease commonly attack the base of the skull; consequently the temporal bone is frequently involved. Initial symptoms of the disease therefore may be aural disturbances. As the lesion of the temporal bone grows in size it may manifest itself in several ways: (1) by perforation of the external wall of the auditory canal; (2) by erosion of the cortex of the mastoid process or of the zygomatic or the squamosal portion of the temporal bone; (3) by invasion of the labyrinthine capsule; (4) by involvement of the facial nerve; (5) by encroachment on structures in the jugular foramen; (6) by secondary infection.

Granulations appearing in the canal of the external ear in the presence of a destructive lesion of the mastoid process and an intact tympanic membrane are suggestive of this disease. The diagnosis can be made by microscopic examination of this granulation tissue.

Secondary infection frequently occurs in such a diseased temporal bone. In that case the underlying disease is often overlooked. An important clue is the

roentgenographic evidence of a destruction of bone out of all proportion to that which one would expect from the duration and the degree of infection. When the diagnosis is suspected, it is important to seek roentgenographic evidence of other skeletal lesions.

Eosinophilic granuloma is a benign form of the disease which manifests itself by one or a few skeletal lesions. The skull is a common site for solitary lesions. As in Hand-Schüller-Christian disease, there is usually little accompanying pain. The diagnosis of Hand-Schüller-Christian disease and eosinophilic granuloma must be made by microscopic examination of involved tissue, as there is nothing about the roentgenographic or clinical pictures which will differentiate these diseases from each other or from several other conditions.

The histopathology of the disorder is basically that of an inflammatory histiocytosis. In Letterer-Siwe disease there is marked proliferation of histiocytes in involved bone and many viscera, with varying degrees of superimposed acute inflammatory reaction. In Hand-Schüller-Christian disease the lesions have accumulations of histiocytes, which are usually filled with lipoid material. The lesions of eosinophilic granuloma are characterized by sheets and strands of histiocytes throughout, which are conspicuous collections of eosinophils. The individual lesions of these diseases respond well to roentgen ray therapy. After the diagnosis has been established by biopsy, roentgen ray therapy should be instituted.

Mastoidectomy should be done only to treat temporal bone lesions which are infected, or when it is necessary to obtain tissue for biopsy. In cases of these diseases mastoidectomy wounds heal slowly unless followed by roentgen ray therapy. Mastoidectomy was performed in 2 of 3 cases of Hand-Schüller-Christian disease of the temporal bone. In 1 the diagnosis was established by microscopic examination of granulations appearing in the canal of the outer ear. The region of the frontal bone immediately above the lateral aspect of the supraorbital ridge appears to be a common site for solitary eosinophilic granuloma. In 1 patient a chocolate-colored cyst with granulation tissue walls was encountered in that area. Such a lesion may represent an eosinophilic granuloma in a late phase of central degeneration, hemorrhage and scarring. Another patient had a large eosinophilic granuloma of the mandible.

All these solitary lesions were removed surgically. Roentgen ray therapy is an equally successful treatment. Common procedure is to establish the diagnosis by frozen section biopsy and to curet away the lesion during the same operative procedure. Should other skeletal lesions appear they can be treated with roentgen radiation.

DISCUSSION

DR. H. B. PERLMAN: From a purely clinical standpoint experience emphasizes that it is easy to overlook the true nature of the lesion, especially when infection is superimposed. This was true in all the cases involving the ear which have come to my attention. The first 2 patients were operated on, and some of the removed material was examined histologically without anyone's recognizing the underlying disease process. The original impression was that of ordinary inflammatory granulation tissue; only after other lesions of bone developed was the original lesion of the mastoid process suspected. Then a careful search through all the histologic material removed at the time of operation revealed the characteristic picture in areas not directly involved in the acute inflammatory response. This indicates that in the presence of an infected lesion all the tissue removed must be carefully examined, preferably by a trained pathologist who is familiar with the histologic variations in different phases of this disease. The fact that the original lesion of the ear may have healed and that the patient may have been discharged

before a lesion develops elsewhere in the body also contributes to inaccurate diagnosis. Considerable time may elapse before a new lesion develops, and the patient then seeks relief of shoulder pain, for example, because of a lesion of the scapula. Superimposed infection of a lesion, especially when complications are present as in the first 2 cases in which the mastoid process was involved, requires complete surgical exenteration.

In the presence of noninfected lesions, however, as in the third case in which the ear was involved, complete operative removal is contraindicated and can be prevented if tissue is available for histologic examination. Involved tissue may be obtained easily if the lesion is in the canal of the external ear, as in one case; in other cases biopsy of the mastoid process may be required. This lesion is sensitive to roentgen irradiation and begins to heal shortly after administration of a small dose. This response can be helpful for differential diagnosis. Even the noninfected lesions may produce a picture resembling that of simple suppuration. Thus, a youngster brought in because of a discharging ear was treated as having simple suppurative otitis both by the pediatrician and by the otologist for several weeks. Recurrent aural granulation and a full red drum membrane without landmarks were apparently compatible with such a diagnosis. However, careful examination while the patient was under anesthesia for removal of the granulation tissue failed to reveal perforation of the drum. The source of the granulation tissue was found to be a defect in the floor of the bony canal just external to the drum. The opposite drum membrane was full, red and devoid of landmarks, but there was no discharge or granulation tissue in that canal. Roentgenograms of the mastoid processes were made and were reported by a junior roentgenologist as showing simple bilateral clouding of small cells of these processes. Actually they showed such extensive change in density far beyond the limits of both mastoid processes that it was considered as normal background, against which the involved dense bone of, and adjacent to, the capsule was viewed. Of course, it is easy to look back after the diagnosis is made and point to the features of the case which set it apart from an ordinary case of otitis media. Insidious, painless onset and recurrent abundant granulations are significant.

Many diseases have to be considered in differential diagnosis. In a child recently examined, extensive destruction of the base of the mastoid bone in relation to the jugular foramen with paralysis of the seventh, tenth and twelfth nerves was due to a nerve cell tumor. A biopsy could be made through the floor of the external canal, into which the tumor had grown.

The whole group of apparently related diseases considered in this presentation is poorly understood. They appear to be, at least in part, concerned with disorders of fat metabolism. Enzyme systems of the cell controlling this fat metabolism are probably involved. Perhaps some of the newer histologic technics for demonstrating the effect of some of these cell enzymes may throw light on this aspect of the problem.

However, there are features that suggest that one is dealing with an infectious process. Further information might be obtained by exhaustive study of the material removed from a noninfected lesion such as one sees in the calvarium or near the frontal sinus. Complete aerobic and anaerobic cultures should be made of this material and it should be inoculated into eggs and into the anterior chamber of the eye. There should also be preparations of sterile extracts for skin testing. Any one preparing to operate on such a lesion should mobilize the resources of the departments of bacteriology and pathology for this type of study.

DR. LAWRENCE J. LAWSON: I wish to present briefly the record of a woman aged 51; the progress of the disease is readily shown in the seven slides presented.

The right eye was glaucomatous and had a cataractous lens, so that it was impossible to see the fundus. The left fundus was anemic, and there were several episodes of dimming of vision. This was followed by complete blindness and atrophy of the optic nerve. There was complete loss of hearing in the right ear and about 80 per cent loss in the left ear. The patient's adherence to Christian Science interfered with observation and therapy.

The seven slides demonstrate the progress of resorption of bone in the pituitary region and the right temporal bone in a typical instance of Hand-Schüller-Christian disease. The first slide shows marked thinning of the posterior clinoid processes. In the following six slides, progressive destruction of the sella turcica, the clinoid processes and the posterior walls of the sphenoid sinuses is seen. Currently there is progressive resorption in the right temporal bone.

There was confusion in the early stages regarding the diagnosis. It was first believed to be a pituitary adenoma. The right eye was glaucomatous, and a consulting ophthalmologist strongly considered the presence of intraocular tumor with intracranial extension. Another consultant felt that it must be an intracranial extension of a nasopharyngeal transitional cell carcinoma. High voltage roentgen ray therapy was given. The patient has remained well nourished. She is blind and deaf. No recent roentgenograms are available at this time.

Dr. Elmer W. Hagens: I should like to record the case of a child 22 months of age who was brought into the clinic at Northwestern University Medical School with a history of recent indefinite discharge of one ear. A large polyp was found blocking the external auditory canal making it practically impossible to see the drum. A roentgenogram showed a definite punched-out area in the region of the mastoid process with the report that, while the roentgenologist was not sure of its significance, the observation was unusual. It was treated as mastoiditis, and at operation it was decided to take out the polyp first. This growth proved to originate on the posterior wall, and the drum looked intact. One pediatrician had said that he incised the drum. After removal of the polyp, when the mastoid process was examined, no cortical bone was found; there was a grayish white tumor, which was enucleated as carefully as possible. The bone over the sigmoid sinus was gone, also that of the posterior wall of the external canal down to and including the anticus tympanicus. The tissue apparently went down to the seventh nerve, but a line of cleavage was made superficial to the canal of the facial nerve. Most of the tissue was removed, leaving a little over the sigmoid; the sinus was normal except where the tumor was attached. The wound was closed and healed well and promptly; facial paralysis developed the next day, which has now pretty well cleared up.

Our pathologist at Wesley Hospital has written an article on eosinophilic granuloma, and he believes that is what was present in this case. We hope the child will get along with roentgen ray treatment. This is the only lesion that is apparent. We were glad we did not go too close to the facial canal, because apparently the tissue was in contact with the facial nerve. No particular infection was noted; the polyp was apparently part of the process which had grown through into the external canal. Bacterial studies were not significant, and when we considered them with the pathologic diagnosis we felt that the area was not infected. It is easy to assume that one is dealing with mastoiditis, especially if a polyp is present, and yet such cases can prove to be cases of tumor.

Dr. M. A. Glatt: About four years ago I presented a case of xanthoma–lipoid granuloma of the temporal bone before this society and discussed the difficulty of preoperative diagnosis when the symptoms involve only the temporal bone. In the presence of suppuration such a lesion often resembles Pneumococcus type III

infection. If suppuration is absent, it is difficult to differentiate the lesion from cancer, tuberculosis or other types of bone disease.

In the young, when the lesion of the skull has been associated with signs pointing to involvement of the pituitary gland, it has been known as Hand-Schüller-Christian disease. None of these authors had a knowledge of the pathologic aspects of the disease. Fundamentally, it is a disturbance of the fat metabolism in the affected reticuloendothelial cells which form the granuloma and is not caused by a disturbance of the general fat metabolism. Such a disturbance can involve any organ of the body. Customs and traditions are not easily discarded, and thus it becomes difficult to dissociate xanthoma of the skull from Hand-Schüller-Christian disease even though the pituitary symptoms, the most important, are absent.

The essayists are fortunate in having had the opportunity to see such a large number of cases, as they are extremely rare in ordinary practice. In presenting the first phase of the disease as eosinophilic granuloma they appear to have given a study of an extremely early stage, which possibly presents few symptoms. However, the later phases, as described by them, and the final phase of fibrosis seem to add to the confusion already existing in classification. I should like to see it more clarified and simplified.

DR. HAROLD F. SCHUKNECHT (closing): In all the cases of eosinophilic granuloma the lesions were treated surgically; roentgen irradiation is an equally good treatment. Common procedure is to establish diagnosis by frozen section biopsy and to curet away the lesion at the same operation. Lesions which develop subsequently can be irradiated.

Dr. Lawson's patient with an extensive lesion of the base of the skull probably had Hand-Schüller-Christian disease. The term "eosinophilic granuloma" should be reserved for those lesions having a milder course and consequently a better prognosis.

A monosymptomatic early stage of Hand-Schüller-Christian disease has been known to exist for many years. Several reports on solitary granulomas appeared in 1940, and since then the descriptive term "eosinophilic granuloma" has been in common use. In 1942 the relationship of these granulomas and Hand-Schüller-Christian and Letterer-Siwe disease was emphasized by several investigators. In Dr. Hagen's patient there was a destructive lesion of the mastoid process, an intact tympanic membrane and granulations in the external canal. Two of my patients presented similar findings. Hand-Schüller-Christian disease is always suggested by this clinical picture. The bony facial canal is often eroded away in such cases. Surgical treatment of the mastoid process in these cases is best directed only at establishing diagnosis by biopsy or at treatment of complicating infections. Complete curettage is hazardous; roentgen ray therapy is the treatment of choice.

There is nothing about the roentgenographic appearance of the lesions of eosinophilic granuloma or Hand-Schüller-Christian disease which will differentiate them from each other or from several other conditions. In differential diagnosis one must consider primary and metastatic carcinoma, primary and secondary cholesteatoma, osteomyelitis, multiple myeloma, tuberculosis, syphillis and fundus lesions. I believe, Dr. Glatt, that there is fairly universal agreement that these diseases are all manifestations of a common disorder. Improvement in terminology probably must wait until the cause is discovered.

News and Comment

ANNUAL MEETING OF OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The tenth annual spring postgraduate convention will be held in Portland, June 19 to 24, 1949. Another fine program has been arranged by the Oregon Academy and the University of Oregon Medical School. There will be four guest speakers—men outstanding in their respective fields: Dr. Lawrence R. Boies, professor of otolaryngology, University of Minnesota Medical School, Minneapolis; Dr. Leland Hunnicutt, associate clinical professor of otolaryngology at the University of Southern California, Los Angeles; Dr. James H. Allen, professor of ophthalmology at the Iowa State University School of Medicine, Iowa City, and Dr. Edmund B. Spaeth, professor of ophthalmology at the University of Pennsylvania Graduate School of Medicine, Philadelphia.

There will be lectures, clinical demonstrations and ward rounds.

Preliminary programs will be out about May 1. They may be secured, with further information, from Dr. David D. DeWeese, secretary, 1216 S.W. Yamhill Street, Portland 5.

IMPORTANT: In order that the course may be made more personal and practical, registrations will be limited to 125.

ANNUAL MEETING OF PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The annual meeting will be held on April 22, 23 and 24, 1949, at the Penn Harris Hotel, Harrisburg, Pa.

Among the speakers who will present papers at this meeting will be Drs. A. D. Ruedemann, J. R. Lindsay, R. O. Rychener, C. S. Nash, J. G. Linn, Arno Town and G. M. Coates.

In addition to formal presentations, the meeting will include a round table discussion on "Headaches." Among the discussers will be representatives of all major specialties. There will be a study club to discuss "Problems of Cataract," and among the discussers will be Drs. Ruedemann, Rychener and Town.

The incumbent officers are: James J. Monahan, president, Shenandoah; Daniel S. Destio, president-elect, Pittsburgh, and Benjamin F. Souders, secretary, Reading.

Directory of Otolaryngologic Societies *

INTERNATIONAL

Fourth International Congress of Otolaryngology

President: Dr. V. E. Negus, London, England.
General Secretaries: Dr. F. C. W. Capps and Dr. W. A. Mill, 45 Lincoln's Inn Fields, London, W. C. 2.
Place: London. Time: July 18-23, 1949.

Second Pan-American Congress of Oto-Rhino-Laryngology and Bronchoesophagology

President: Prof. Justo Alonso.
Secretary: Dr. Chevalier L. Jackson, 255 S. 17th St., Philadelphia 3.
Place: Montevideo. Time: January 1950.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Henry B. Orton, 224 Delavan Ave., Newark, N. J.
Secretary: Dr. J. Milton Robb, 1553 Woodward Ave., Detroit, Mich.
Place: Atlantic City, N. J. Time: June 6-10, 1949.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Conrad Berens, 35 E. 70th St., New York.
President-Elect: Dr. J. Mackenzie Brown, Box 584, South Laguna, Calif.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

American Broncho-Esophagological Association

President: Dr. Paul Holinger, 700 N. Michigan Ave., Chicago 11.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore.
Place: Drake Hotel, Chicago. Time: April 18-20, 1949.

American Laryngological Association

President: Dr. Frederick T. Hill, 177 Main St., Waterville, Maine.
Secretary: Dr. Louis H. Clerf, 1530 Locust St., Philadelphia 2.
Place: Biltmore Hotel, New York. Time: May 16-17, 1949.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. John J. Shea, 1018 Madison Ave., Memphis, Tenn.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Drake Hotel, Chicago. Time: April 18-20, 1949.

SECTIONS:

Eastern.—Chairman: Dr. Harold G. Tobey, 403 Commonwealth Ave., Boston.
Southern.—Chairman: Dr. Watt W. Eagle, Duke University, Durham, N. C.
Middle.—Chairman: Dr. Dean M. Lierle, University Hospital, Iowa City.
Western.—Chairman: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.

American Otological Society

President: Dr. Marvin F. Jones, 121 E. 60th St., New York.
Secretary: Dr. Gordon D. Hoople, Medical Arts Bldg., Syracuse 3, N. Y.
Place: Biltmore Hotel, New York. Time: May 18-19, 1949.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Samuel F. Kelley, 47 E. 61st St., New York 21.
Secretary: Dr. Norman N. Smith, 291 Whitney Ave., New Haven 11, Conn.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Rea E. Ashley, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

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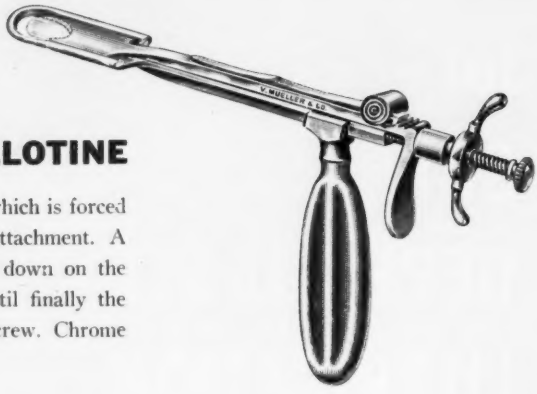

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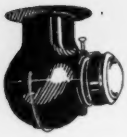

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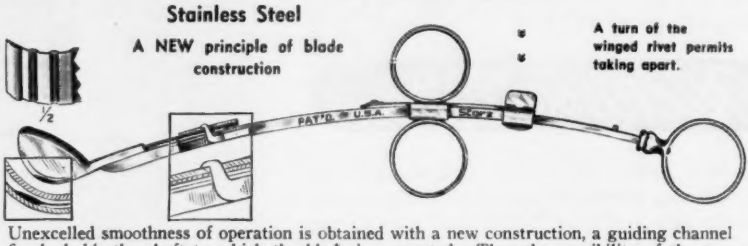

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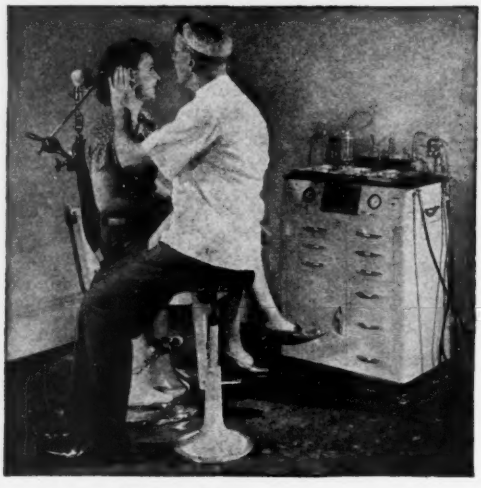

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